Depressive equivalents

by J. J. López Ibor

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General considerations

The introduction of the two concepts "masked depression" (also referred to as "hidden depression", "missed depression", etc.) and "affective or depressive equivalents" (which I called "thymopathic equivalents" in my book on "Vital anxiety" that appeared in 1950[39]), as well as the classification which Kielholz[36] published some time ago and which, by virtue of its clarity, should constantly be borne in mind, have greatly widened the approach to depressive illness and its treatment.

Masked depression is something similar to what the classic psychiatrists termed *depressio sine depressione*—that is to say, it is a condition in which the patient himself does not experience depression or sadness as an important symptom of his disease.

It should be pointed out in passing that, for reasons well known to all of you here, the distinction between endogenous and reactive depression is not as well documented as it might be. Various attempts to justify this distinction by reference to statistics have not proved entirely convincing, nor have deductions based on genetic studies, symptomatology, and response to treatment. A vast number of papers have been published claiming that electroshock treatment, at the time it was first introduced, produced very beneficial results in many patients diagnosed as suffering from reactive depression; in fact, the proportion of good responses was similar to that obtained in endogenous depression. Nevertheless, this problem, which is much further from being solved than one might think, is not what I want to talk about today. I should like instead to concentrate on the subject of so-called *equivalents*. The word "equivalents" has been used in various medical contexts, one of the most important and recent of which is epilepsy. It has indeed been demonstrated not only by clinical research but also by electro-encephalographic recordings, pathologico-anatomical studies, and therapeutic trials that these epileptic equivalents do exist.

The existence of depressive or affective equivalents, on the other hand, seems more difficult to establish. And yet, if we consider the *ictal neuroses* described by Jonas[34], for example, and if we analyse the case histories in his book, we are bound to conclude that these ictal neuroses have nothing to do with epilepsy but, on the contrary, fall under the heading of depressive illness.

The study of depressive equivalents should not be limited simply to isolating somatic symptoms which may accompany depression or anxiety. The somatic symptoms of *anxiety* have been dealt with at length by Freud[25] and also by

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STEKEL[64], whereas those of depression have been studied by the authors of classic medical treatises and also, in greater detail, by more recent investigators in the field of depressive illness.

Neuroses and depressive equivalents

It is customary to define neuroses as disorders due to the inward processing of experiences. KURT SCHNEIDER declared that "an experiential reaction consists in a motivated and meaningful response to an experience". To these two definitions we might add numerous others, all revolving about the same axis. The problem is to draw the line between a normal response and a response that exceeds normal limits. The response may be abnormal in respect of the somatic manifestations to which it gives rise or simply in respect of its violence. What distinguishes a normal from an abnormal reaction to experience has been clearly pointed out in phenomenological terms by JASPERS[33] *.

Long experience has shown me that neurotic disorders display an *endothymic structure* similar to that observed in mild forms of depression. So-called *vital or endothymic sadness*, as well as *vital or endothymic anxiety*, may be encountered in both neuroses and depressions; in both cases they may exhibit the same basic features, even though they may differ in intensity and in the form they take.

The anxiety of a neurotic, or the sadness of a depressive, is not the same as that of a healthy person. Consequently, as I have already said on previous occasions, I am inclined to believe that the abnormalities met with in neuroses or in psychosomatic disorders are referable to the patient's state of mind or mood.

Anxiety plays a predominant role in neuroses. FREUD himself said that anxiety was a cardinal feature of neuroses, but this anxiety of the neurotic, just like the sadness of the depressive, differs from the anxiety or depression of a normal subject. In a normal subject, the psychic reaction of the ego to an experience is commensurate with the actual experience itself.

Coenaesthesia is a term coined by physiologists and psychologists in order to express the idea that messages are transmitted from each organ, or from each cell, to the centre of the personality, i.e. to the so-called ego, by a mechanism resembling an electromagnetic wire. We are accustomed to thinking in mental clichés which do not in fact correspond to reality, although, of course, it is true that this reality is often extremely difficult to apprehend. Characteristic of all mental illnesses is the peculiar sensation the patient has that his physical or somatic symptoms are dissociated from his psychic disturbances. The ego seems to disintegrate to some extent. The normal subject, however, feels that he is unable to control his body without attributing a certain meaning to it, a meaning which is dictated by the inapprehensible centre we call the "ego". The significance of the ego, like the significance of life itself, only becomes apparent in the course of life and even then not completely. Hence, the relationship between the soma and the psyche, as conceived of by psychologists and psychiatrists, is really based on a false premise.

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* KURT SCHNEIDER[56] later submitted these considerations to a critical assessment.

HARTMANN[31] used the term "metaphysical" to denote those problems which are beyond our comprehension, and the relationship between the soma and the psyche is probably just such a problem.

Words such as "psychogenesis" or "conversion reaction" may well facilitate the description of a disorder, but at the same time they act as a cloak that conceals the reality from us. We might therefore be justified in introducing an additional term—i.e. "*catagenesis*"—to describe cases in which a disorder seems to be due to a psychological trauma, but manifests itself days or weeks afterwards in the form of an organic disease. Examples of this kind have been quoted in the literature, and I myself have mentioned quite a few in my publications.

The body is an instrument of the ego and thus, like the ego, it possesses an intentional character. Although the Cartesian approach has been of great assistance to medical science in many respects, it has also diverted attention from the real essence of the question. It is true that the concept of psychosomatic disorders has opened up new perspectives in the investigation of certain clinical problems, but this concept, in its turn, will have to be submitted to re-interpretation. One possibility is to include many of the disturbances described as psychosomatic under the heading of mild or vegetative depression. In cases of "syndrome shift", for example, certain disorders generally held to be psychosomatic alternate with others which, even to the most sceptical of investigators, are obviously of a depressive type.

The depressive nature of these disorders is borne out in many cases by their response to treatment. A large number of patients suffering from psychogenic illnesses respond to purely psychological treatment, i.e. to psychotherapy alone. When we analyse these cases, however, we are often struck by the strange fact that psychotherapy usually has to be given for the entire duration of the disorder—or, to be more accurate, for the length of time the disorder could be expected to persist on the basis of clinical experience.

There are many other examples which could be quoted, but my intention here is merely to draw attention to this phenomenon and to suggest that it should be examined in greater detail. We should not forget that, far more often than is generally supposed, illnesses which used to be referred to at the turn of the century as hysteria, neurasthenia, etc. are associated with localised symptoms which should be classified among the depressive equivalents.

Masked depression and depressive equivalents

When talking to patients and also when writing in medical journals, we now prefer the term "depression" to "melancholia". There are several reasons for this: it is not simply that we are trying to avoid the historical connotations of the word "melancholia" or use of the expression "manic-depressive illness". It is because a far higher proportion of the cases seen nowadays in out-patient departments and hospitals display these diseases in a milder form. Nevertheless, the meaning of the term "depression" has to be carefully defined. Many authors, influenced by the ideas of FREUD, are accustomed to think that depression is due simply to the real or imagined loss of the "object" on which the ego libido

is concretely or symbolically dependent. The feelings of despair or of hope that commonly occur at the start of a depressive phase are assumed to be merely evidence of the patient's surrender to or struggle with his sense of loss.

There is no need to point out that in everyday language we use words such as *pain*, *anxiety*, *depression*, and *sadness* as if they referred to clearly separate concepts, as if they were unconnected islands. The most that some of us would be prepared to concede is that these islands may be regarded as forming an archipelago.

In their book *Wesen und Bedeutung des Schmerzes* ("Nature and significance of pain"), published in 1936, SAUERBRUCH and WENKE[54] refer mainly to developments in the field of psychology during the previous 25 years. In view of the many different definitions and interpretations of pain, however, WENKE himself began to wonder whether pain should be regarded as a sensation or as a feeling *(Empfindung* or *Gefühl)*. He believes that pain should be termed a "sensation" only when it is experienced in a *neutral* sphere of consciousness, and as a "feeling" when it is directly related to an attitude of mind and even induces an action. As BERGSON[7] said, "every pain, therefore, consists of an effort and of an impotent effort at that".

On the other hand, we know full well that so-called *causalgiform* pain includes painful disorders which differ qualitatively from causalgia itself. The physiologist ACHELIS[1-3] sums this up in a phrase reminiscent of the sentence from BERGSON which I have just quoted: "Pain would then no longer be a sensation, but the experience of an effort on the part of the organism."

Let us now pass on to another problem: in psychiatry the attempt is made to separate anxiety from depression and even to distinguish—with complete justification from the clinical point of view—anxious depression from non-anxious depression. And yet, if we cease for a moment to think in terms of sadness and concentrate instead on the word "melancholia", we shall see that melancholia, like anxiety, comprises three basic states of mind: the two poles of religiousness and nihilism, a feeling of impoverishment, and a feeling of physical disease (hypochondriasis). It would take too long here to discuss in detail the transitions that occur between these various states of mind, but it is essential to recognise that such transitions are perfectly real. What is artificial—though useful at times—is the radical separation of these states of mind into three distinct categories.

In depression, vital or endothymic feelings—including, in particular, normal sadness—are, as it were, extinguished. A patient suffering from depression does not need to have lost anything; he may simply have experienced a pathological lowering of his vital mood.

Consequently, it is not only legitimate but also essential to use the terms "masked depression" and "depressive equivalents".

Neurological signs and symptoms masking depressive equivalents

In this paper of mine I shall confine myself to a particular group of depressive equivalents which are frequently encountered behind a mask of neurological signs and symptoms.

It is my belief that so-called psychosomatic disorders are in many cases depressive equivalents. These disorders include, for example, various types of headache, to which I shall not refer here because I dealt with them at last year's symposium in St.Moritz[42].

Akathisia

Of all the disturbances which I regard as depressive equivalents, one of the most interesting, in my opinion, is *akathisia*, which was described by BING[9] in 1923. This akathisia may either take the form of a muscle spasm in a patient who is otherwise exceptionally calm, or else it may force the patient to keep constantly on the move, extending and flexing his limbs without respite. DELAY and DENIKER[13] draw a distinction between akathisia, which they define as an inability to remain quietly lying down, and *tasikinesia*, which is a compulsive need to keep moving about. Both terms, however, denote the presence of uncontrollable locomotor activity, sometimes accompanied by dysaesthesia. The disorder may occur spontaneously in the course of a disease such as parkinsonism, or else it may be induced by drugs—e.g. by major tranquillisers used in the treatment of certain psychoses. It should be noted, though, that spontaneous akathisia was already known long before the advent of the major tranquillisers; it can develop in any patient for no apparent reason and is probably due to a disturbance in nervous regulatory mechanisms.

The reason why I have chosen akathisia as an example is to point out right away that some of the disturbances I shall be mentioning may in fact be attributable in certain cases to organic lesions or diseases which have nothing to do with the category of depressive equivalents. When faced with a patient displaying one of these syndromes, the physician has to learn how to distinguish between an organic and a non-organic aetiology. This is not an easy task, because in both cases the pathogenesis may be largely similar.

"*Restless legs*"

In recent years many papers have been published on the disturbance generally referred to as "*restless legs*". Of the numerous synonyms used to describe this clinical entity I shall mention only the following: anxietas tibiarum, Ekbom's syndrome, Wittmaack-Ekbom disease, nocturnal paraesthesia of the lower limbs, leg jitters, and fidgety legs.

This syndrome was described as far back as 1685 by THOMAS WILLIS[71] who said that some people, when in bed at night, experienced tendon contractions in their legs and such a pronounced "tossing" of the limbs that they felt as if they were in a torture chamber and could not sleep.

For a long time the term most commonly applied to this syndrome was "anxietas tibiarum"; WITTMAACK[72] believed it to be a sign of hysteria. BEARD[5], in his book on neurasthenia published in 1890, likewise spoke of patients who were unable to keep their legs still in bed at night because of a sensation which gradually developed into an intolerable pain. These patients had to get up and walk about, even though the exercise made them feel weaker and aggravated their symptoms. BEARD regarded the syndrome as one of the many consequences

of spinal irritation. Oppenheim[50] included restless legs under the heading of neurasthenia; describing the disorder, he said that the patients experienced a tormenting sensation in the legs which could last for years or even decades, and he suspected that heredity might play a part in the aetiology of the syndrome. Many other authors have studied this problem, including in recent years Ekbom, who has published a number of outstanding papers on the subject[15-22].

The most important single symptom of restless legs is *dysaesthesia* which is occasionally confined to the region of the knee, but often spreads downwards as far as the feet. As a rule, it is bilateral and symmetrical, but it may affect one leg in particular or alternate from one leg to the other. The patient complains that the sensation is not on the surface of the leg, but deep down inside it; this explains the reference to the bones in one of the designations for the syndrome already mentioned (anxietas tibiarum). It is rarer for similar sensations to be experienced in the arms and hands at the same time, but only a few days ago I came across a 60-year-old man in whom these sensations persisted throughout the day and spread to his entire body, with the result that he could not remain sitting or standing but had constantly to move from side to side. His tormenting symptoms, which he found very difficult to describe, were reminiscent of akathisia.

Many patients talk of feeling an itch inside their limbs, some have the impression that their feet are about to go to sleep, and others refer to a diabolical sensation akin to torture. They are often virtually unable to give an exact description of their symptoms. Oddly enough, as in *nocturnal brachialgia paraesthetica*, these symptoms almost invariably disappear when the limb is moved, and they occur for the most part in bed at night. In milder cases, the patient falls asleep after half an hour or an hour, especially if he has taken a sedative or hypnotic; he may, however, wake up again after an hour or two because the sensations return. In my experience, some of these patients try to obtain relief by walking over cold surfaces—a tiled floor, for example—and they avoid warmth. One author, incidentally, quotes the case of a woman who used to dance the Charleston before going to bed! The disorder is of course accompanied by insomnia, and even when the patients do fall asleep, they sleep only lightly, with the result that their daily life is adversely affected and, as many of them say, their sense of well-being is destroyed.

In some patients the restlessness seems to disappear if they contract a disease associated with high fever. Others complain that their sensations are really painful, very similar in fact to what they feel when they have a tension headache. In about 5 % of cases, the sensations are only mild, but as a rule they are extremely severe. This phenomenon of restless legs has been attributed to a variety of causes. Wittmaack believed the syndrome to be inherited; Ekbom likewise refers to hereditary and familial factors of a dominant type and quotes the case of monozygotic twins whose mother also displayed the same syndrome. Other authors, too, such as Oppenheim, whom I have already mentioned, as well as Mussio Fournier and Rawak[47], have observed that the syndrome can be hereditary.

In 1951 Ekbom[19] noticed that the syndrome improved in patients given an iron preparation because of anaemia, and therefore he, as well as Nordlander[49],

attributed restless legs to sideropenia. These authors found that the syndrome was associated with reduced serum iron levels (60 mcg. per 100 ml.). Some investigators have found a decrease in haemoglobin, and others sideropenia accompanied by a haemorrhagic peptic ulcer.

In the course of a study conducted in gastric ulcer patients in a surgical ward, a collaborator of mine determined the serum iron levels before and after operation. He encountered only two cases of restless legs in this group; some of the patients did in fact have sideropenic anaemia, but he was unable to find any correlation between this and restless legs. This syndrome has also been reported very occasionally in cases of malignant diseases, such as carcinoma.

In 1953 TEN BERGE[6] stated that he had observed the syndrome of restless legs in 27 % of pregnant women; it appeared during the second half or last three months of pregnancy, and he likewise attributed it to an iron deficiency. In this connection, I should like to draw attention to the occurrence, in some pregnant women, of "burning feet", caused by a deficiency of pantothenic acid. When pellagra was rife in Spain following the Civil War, I saw many cases in which all the symptoms of this disease, with the exception of "burning feet", responded to treatment with the usual anti-pellagra vitamins; the "burning feet" could only be relieved by administering pantothenic acid. I have also observed this symptom in women in an advanced stage of pregnancy. In these cases, the sensation of "burning feet" promptly disappeared in response to the intravenous administration of pantothenic acid, and therefore had no connection with the phenomenon of "restless legs". It occurred in women who had insisted on keeping to a diet, even during pregnancy, because they did not wish to put on too much weight. The presence of "burning feet" has also been reported in infectious diseases and in certain metabolic disorders, and in these instances it has been attributed to an increase in serum cholinesterase.

As regards the psychological influences involved in the "restless legs" syndrome, horror films, for example, have been indicted as a causative factor, and some of EKBOM's patients said that they could not go to the theatre, cinema, or concerts without these sensations developing. WITTMAACK, BEARD, and OPPENHEIM state that, whereas some patients with restless legs may feel slightly depressed or anxious, the majority are perfectly happy and well balanced and display no signs of mental disturbance. This is also the opinion of SELVAAG[60]. Nevertheless, the view that the syndrome is of hysterical origin is so widespread that patients may be afraid to mention it unless the symptoms are very pronounced.

Brachialgia and meralgia

In numerous types of *brachialgia* the somatic origin of the pain is both clear and defined, but there are also forms in which the origin is not so evident. It was WARTENBERG who long ago discovered the syndrome which he referred to as *static brachialgia paraesthetica*[65]. Briefly, this is a syndrome which appears in one or both arms when the patient wakes up in the morning; unlike other types of neuralgia it disappears only in response to movement.

It was thought initially that in many cases the pain was simply due to compression of the arm resulting from an awkward sleeping posture, but subsequent

experience has shown that this cannot be the full explanation. Brachialgia paraesthetica is associated with dysaesthesia, affecting especially the hands, in which, moreover, genuine acroparaesthesia may develop.

In his first description of the syndrome WARTENBERG laid great emphasis on this point—that is, on the purely mechanical element of compression. But in his book on "Neuritis, sensory neuritis, neuralgia", which he published after he had gone to the United States[68], he attributed far more importance to somatic factors. He even called the syndrome a *brachial neuritis*, as if it were due to inflammation of a nerve. There is, of course, no evidence to support this assumption, and even WARTENBERG himself had previously refused to consider it as a possibility. WARTENBERG also described a *cutaneous neuralgia affecting the dorsal surface of the forearm* which could not be attributed to trauma, overstrain, or any other definite cause. This syndrome belongs to the same category as *cheiralgia paraesthetica.*

In many of these types of neuralgia which he now included under the heading of "sensory neuritis" WARTENBERG found that zones of hypaesthesia and hypalgesia existed side by side with zones of greater sensitivity; this is difficult to account for if the syndrome were really due to organic nerve lesions.

In many treatises on neurology—as, for example, in STAAL's recent contribution[63] to VINKEN and BRUYN's "Handbook of clinical neurology", *meralgia paraesthetica* still figures among the diseases caused by nerve compression.

KLIMKE[37] believes that the commonest site for this compression is the inguinal ligament, but he also mentions the lower part of the iliopsoas muscle below the aponeurosis and the area in which the nerve leaves the muscle. BOLLINGER[11] stresses the fact that although this nerve runs close to the caudal pole of the kidney, passing behind the caecum on the right side and below the descending colon on the left, he did not encounter any case of meralgia paraesthetica among 158 patients suffering from renal tumours. He also points out that the site of compression might be in the tendons of the oblique and transverse abdominal muscles or in the aponeurosis. Finally, WARTENBERG[67] states that the compression can occur at any point in the course of the nerve from the thalamus to the periphery.

The patient complains of paraesthesia, of numbness and pain, of hypaesthesia or anaesthesia, affecting especially the zone innervated by the anterior branch of the external cutaneous femoral nerve. Some say that the feeling is akin to a burning sensation. The disorder is seldom bilateral. Sometimes the patient declares that he can hardly bear to have any clothing touching his skin at the affected site and that the symptoms become worse when he walks. Neurological examination may reveal various abnormalities in the areas served by the anterior branch of the nerve. ECKER and WOLTMAN[14] point out that sense of touch, as well as sensitivity to pain or heat, are reduced in 60% of patients, but that no patient ever makes a mistake in evaluating the amount of pressure exerted on the area affected. Some patients indicate a trigger point located along the antero-superior iliac crest. BOLLINGER[11] has described the presence of trophic changes, such as hypotrichosis, in some instances, and CHHUTTANI et al.[12] even observed atrophy of the quadriceps muscle in 13 cases.

It is worth recalling that FREUD himself suffered from meralgia paraesthetica and that BERNHARDT[8] and ROTH[53] both published their description of the syndrome in 1895. Opinions as to its aetiology are divided. Some, such as MÓRITZ[45], for example, deny that genuine meralgia exists; they believe it to be a symptom either of a vertebral disorder or of a disease process in the abdominal cavity. Other authors refer to hereditary or constitutional factors. GOLDSTEIN[28] encountered meralgia paraesthetica in two young brothers, but no clear proof of a familial predisposition has yet been found. In his study of 158 cases BOLLINGER comes to the conclusion that the disorder may result from fracture of the pelvic bones, from falling heavily on the buttocks, etc. ECKER and WOLTMAN insist that abdominal compression plays an important role, and that this compression may stem from an inguinal hernia. They mention one case in which the meralgia disappeared after the patient had carried a gold watch in his pocket. A host of explanations—most of them resting on very unsure foundations—have been put forward to account for the syndrome, and it has even been alleged that the weight of military equipment might be to blame in some cases.

MÓRITZ believes that the cause of the disorder is located in particular in the vertebral column or in the vicinity of the antero-superior iliac crest, and that it is more likely to develop in the presence of obesity or of a ganglion on the external cutaneous femoral nerve. According to some authors, the possible causes even include spina bifida, sacralisation of the fifth lumbar vertebra, and calcification of the lumbo-sacral intervertebral disc. BODECHTEL[10] describes one case in which the meralgia was due to an aortic aneurysm, and other authors have attributed it to various tumours, such as pelvic sarcoma.

To sum up, almost all neurologists have come to the conclusion that in every case of meralgia paraesthetica, all possible causes, including even micro-trauma due to the clothes the patient wears, must be carefully investigated. The disorder is commoner than might be thought, particularly if one bears in mind not only the statistics quoted by the neurologists, but also clinical experience. As regards treatment, STAAL himself points out that he succeeded in eliminating meralgia paraesthetica in many cases by cooling the skin to the point of anaesthesia, even though he does not deny that resection of the nerve may be necessary in some instances. In one patient with very refractory meralgia paraesthetica HAGER[30] even carried out a resection of the iliac crest, and it is safe to say that the majority of authors are inclined to favour a surgical approach.

It strikes me that the neurologists, perhaps owing to the type of patients they have to deal with, omit to mention a number of facts which seem to me to have an essential bearing on any definition of meralgia paraesthetica. First of all, the spread of the symptoms does not correspond exactly to the distribution of the appurtenant nerve. Secondly, the patients complain not of anaesthesia, but of a peculiar sensation which they find difficult to describe; moreover, it varies so much in that it can appear in one leg on one day and then in the other leg two days later. I was recently called upon to treat a woman whose meralgia paraesthetica had led the surgeons to suspect the presence of a tumour in the lower regions of the spinal cord or in the nerve roots in that area. They carried

out every possible examination, including several myelographies, and eventually decided to administer radiotherapy, because they thought an operation on the spinal cord might be dangerous in view of the patient's age (65 years). She had already become dependent on drugs by the time I first examined her. In another case I know of, an attempt was made first of all to anaesthetise the nerve. The meralgia promptly disappeared for a few hours and the surgeon in charge therefore decided to resect part of the nerve. Within 24 hours after the operation, however, the meralgia returned. In the cases I have seen, meralgia often has this habit of appearing and disappearing suddenly; moreover, the sensation, which the patient is usually unable to describe clearly in everyday words, is similar to that reported in other disorders of the same type. *If we study the patient's past history carefully and keep him under close observation, we frequently discover that he is suffering from an underlying depression and that, once this depression has been properly treated, the meralgia vanishes.*

This does not mean, of course, that meralgia paraesthetica is never caused by lesions of the peripheral nerves. Any neurologist or neurosurgeon will quite rightly declare that he has actually experienced cases in which a nerve lesion was definitely implicated. However, I consider it important to distinguish between the two types of aetiology—traumatic or infective, on the one hand, and depressive on the other.

Migratory neuritis

There is a special form of neuritis which, owing to one of its clinical features, is described as "*migratory*". In my opinion, it is precisely this migratory nature of the disorder that suggests that it should really be included among the depressive equivalents. SCHULTZE[58,59] found that he himself suffered from multiple migratory neuritis. It caused him discomfort, but was not associated with paralysis or paresis. It must not be forgotten that SCHULTZE was the author who described the condition known as acroparaesthesia and that he was consequently very familiar with this type of symptom. It cannot be said that these sensations are all that common, but they do exist and they serve to confirm the thesis put forward in this paper. Cases occur, for example, in which transient neuritis develops in the thigh—that is to say, in the region innervated by the external cutaneous femoral nerve, the same nerve that is affected in meralgia paraesthetica. After a while the neuritis disappears and is superseded by an hypaesthetic area on the dorsal surface of the forearm. This hypaesthesia, too, then vanishes, only to reappear later in some of the fingers, above the right shoulder-blade and, finally, in both feet.

Although the number of cases of migratory neuritis that have been described in the literature is not very large, it is sufficient for us to be sure that the disorder exists.

All these forms of dysaesthesia in various localisations may be accompanied, as I have already said, *by an underlying state of anxious depression* which is not easy to detect unless the physician is on the look-out for it and has a wide experience to help him. The establishment of a correct diagnosis in such cases, however, has a most important bearing on their treatment, because if they are found to be depressive

equivalents, they should be treated as such, and there is then no need to search for any other cause of a vasomotor, infective, or traumatic nature.

The approach to psychosomatic medicine and depressive equivalents

The whole approach to so-called psychosomatic medicine requires radical revision. Everyone will agree that since the Second World War this branch of medicine has acquired tremendous importance and in some places it has even become a discipline in its own right. This, however, is going too far, because the vast majority of patients in this category are really psychiatric cases, inasmuch as they are suffering from masked depression or depressive equivalents. Initially, psychosomatic diseases were regarded as being of psychogenic origin and it was therefore thought that the only way to treat them was by psychoanalysis. It is true that a psychosomatic disorder may respond to psychoanalysis, but this response probably means nothing more than that the psychoanalysis has coincided with a spontaneous remission in the depressive phase.

In my experience, at least, and according to the opinions expressed by many authors in the relevant literature, less importance is now attached to the establishment of symbolic relationships between symptoms and complexes, or to interpretations of a purely psychoanalytical nature. Current views on the subject have been summed up by an American psychoanalyst who devoted most of his time to patients with psychosomatic disorders. I am referring here to GITELSON[27], who died only recently. In one of his last published papers he clearly stated that in the light of his long experience it was impossible to correlate the data obtained from a psychodynamic exploration of the patient with the evolution of that patient's disease. He felt that this approach to the problem should, in fact, be abandoned, and that the physician should devote his efforts in such cases to what has been termed "strengthening the patient's personality or ego".

There can, I think, be no denying that psychotherapy is both useful and necessary in the vast majority of patients with psychosomatic disease, particularly if we employ the word "psychotherapy" in a broad sense. The classic authorities in this field intuitively knew a great deal about psychotherapy. Psychodynamic investigations have added enormously to the literature on the subject, but they have not done much to improve our knowledge. ALEXANDER[4] claimed that he could tell the disease from which a patient was suffering simply by studying that patient's personality. However, if we analyse his results carefully, we will find that they are not particularly convincing. Consequently, psychosomatic medicine has developed along other lines which I do not intend to pursue here, even though some of them are extremely interesting.

One of the most important tasks—not only because it is concerned with clinical realities, but also because its successful solution would greatly facilitate an understanding of how pathological changes develop in many cases—would be to study the course of psychosomatic disorders. If you deliberately keep track of a patient over several years, as many authors, including myself, have done on various occasions, you will be struck by the fact that one disorder may be

followed, after a fairly long interval and without the occurrence of any obviously pathological changes, by another. In my book entitled "Vital anxiety", I referred to this replacement or switching of syndromes. Other authors, such as GROEN et al. [29] have called this phenomenon "syndrome shift". SPIEGELBERG[61, 62], who devoted great attention to this problem, speaks of "syndrome alternation". Whatever name we choose to give it, the fact remains that this appearance and disappearance of a disorder, followed by its reappearance in an apparently different form at some other site, constitutes what might be termed a "pathological metabasis" *. It has been reported, for example, that gastrectomy is often followed by a psychosomatic disorder, the symptomatology of which is no longer localised but generalised. PFLANZ [51] followed up patients who had been subjected to vagotomy because of an ulcer, and he found that when the ulcer symptoms disappeared they were replaced by others; in some cases, even, the same subjective symptoms as the patient had experienced prior to surgery reappeared subsequently although no recurrence of the ulcer could be demonstrated radiographically. He called this syndrome "phantom ulcer pain". I myself have known a number of cases in which patients complained of extremely violent headaches following gastrectomy. The headaches were attributed initially to the anaesthesia, but, as they failed to subside, it was concluded that they must be due to a form of symptom replacement.

In other patients, the time lag between the disappearance of one symptom and the appearance of the next is much longer. By way of example, I can quote the case of a man who, at the age of 25 years, went through a period of very severe insomnia; when he was 30, he developed homosexual phobias which lasted about a year, and at 40 years of age he presented with depression accompanied by syphilophobia. Finally, when he was 45, he had to be operated on for a gastric ulcer.

There are also many cases in which the patient displays an asthma-like syndrome in his youth, and then, 10 or 12 years later, complains of some painful condition, such as coccygodynia or meralgia paraesthetica, which we would classify as a depressive equivalent.

By mentioning these examples, I merely wanted to show you that we have not yet succeeded—and I do not think it likely that we ever shall—in predicting the type of syndrome that may occur or the course it may follow. It seems to me that recent investigations, in which attempts have been made to predict, on the basis of a patient's way of life, the disease he will suffer from in the future, have not proved very convincing. The results obtained are similar to those yielded by studies based on an assessment of the constitutional type to which a patient belongs. Evidently, more extensive and more detailed investigations are needed in this field **.

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*Webster's Third New International Dictionary defines "metabasis" (from the Greek *metabainein* meaning "to change") as "a shift from one subject, point, or division to another; a medical change (as of disease, symptoms, or treatment)".

** MEADE et al.[44], among other authors, have reported a decrease in the incidence of duodenal ulcer in recent times, and MORRIS [46] has observed that the mortality of the disease is also falling.

What is certain is that in the period between the two World Wars great changes occurred in the manner in which diseases presented themselves. Peptic ulcer appeared for the first time at the beginning of this century and attained its highest incidence (in terms of the number of cases diagnosed) between 1930 and 1940. Gastric ulcer was more common among men belonging to the lower social classes. Duodenal ulcer, found most frequently in middle-aged subjects, appeared to show no clear-cut correlation with social class.

During the Second World War, the situation changed, inasmuch as the incidence of gastric ulcer began to decline in 1940. Judging from statistics published in 1968[52], moreover, this decline seems to have become accentuated in recent years. The reasons are obscure. Various explanations of a psychological nature have been postulated, such as changes in the type of stress to which people are exposed nowadays, and it is even probable that in the group of chronic, non-communicable diseases psychological factors related to everyday life do play a dominant role. As KESSEL and MUNRO[35] have said, it is becoming steadily more difficult to establish exact diagnoses and, above all, to determine when a disease is psychosomatic. What is happening, then, in the case of the diseases with which we are concerned here, is the same as what happened with hysteria—i.e. they are changing in both incidence and manner of presentation owing to the age in which we live. At all events, this phenomenon of the "syndrome shift" compels us to consider the possibility that all these various syndromes have a common basis, even though we may be unable to define that basis.

NEMIAH and SIFNEOS[48], as many other authors have done before them, recently stressed that psychosomatic personalities all belong to the same type. They point out that the great majority of such patients are lacking in imagination or in some other equivalent quality of an intellectual or affective nature. However, this suggestion, like previous attempts to explain psychosomatic disorders (by blaming them, for example, on immaturity of the personality, on ego strength or ego weakness, etc.), must be viewed with some scepticism.

It would perhaps be more productive if we were to try to discover whether such diseases occurred in *cycles*—not in fixed cycles, as FLIESS[23] thought and as FREUD accepted for a long time, but in cycles varying from one individual patient to another. Even if this were true, however, we would still be left with the second problem—that of the morphology or type of the disease. This second problem, though, might be easier to solve *a posteriori* by reference to the patient's age, ecological and psychological factors, etc. We should thus have an entirely new approach to psychosomatic pathology, an approach similar to that adopted by psychiatrists when they attempt to predict recurrences in patients suffering from psychoses of a cyclic nature.

For my part, I feel that this is an extremely important and realistic approach to the study of psychosomatic pathology and to psychiatry as a whole. You will perhaps remember the case of FREUD's "Wolf-Man"[26], whose disorders were first attributed to the so-called "primal scene", i.e. to the fact that at the age of one and a half years he had seen his parents having sexual intercourse. When making this deduction, FREUD took no account of the cycles of disease which the patient had had since childhood. At the beginning of the 1914–1918 war, the

patient was discharged as cured. But later on during his long life he was plagued by a series of depressive phases which were the real reason for his disorders. Thanks to the discovery of new facts which have added to our knowledge, attempts to explain psychosomatic diseases by reference to the patient's way of life appear much more interesting nowadays than they did when they were first undertaken—more interesting even than VON WEIZSÄCKER's[69, 70] insinuation that there may be an equivalence between somatic and psychic disorders. I do not wish to imply that all psychosomatic diseases should be classified as depressive equivalents. It is for the moment difficult to put this interpretation on several psychosomatic diseases, such as gastric ulcer or mucomembranous colitis, which have been very extensively studied. On the other hand, agoraphobic vertigo and certain other disorders can quite easily be interpreted along these lines, because they respond radically to antidepressant therapy. I know of five patients in whom vertigo failed to disappear in response to the usual surgical operation, but vanished completely once it had been treated as a depressive equivalent.

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Discussion

G. Nissen: In child psychiatry the problem of the hyperkinetic, hyperactive child plays an important role. In the last ten years or so, amphetamines have come to be used on a much wider scale in this indication—especially in the U.S.A.—as a result of studies on "minimal brain dysfunction" (M.B.D.). We in Europe have so far either rejected this form of treatment or have adopted a wait-and-see attitude towards it. I find it very difficult to account for the so-called paradoxical mode of action of amphetamine in the M.B.D. syndrome. Although amphetamine is not an antidepressant, one wonders whether somatic equivalents in the form of tasikinesia or "restless legs", as described by Dr. López Ibor in connection with adult patients, might not be implicated in some of these hyperkinetic children. In the light of my experience, evidence of severe emotional frustration can be found in a number of markedly hyperkinetic children whose past history contains no record of minimal brain damage during early childhood. In these children we obtain good results by giving them small doses of antidepressant drugs. In my view, there is no doubt that masked depression can also be involved in such cases. Owing to the fact that the symptomatology is less pronounced in children, little progress has yet been made in the study of depressive equivalents in child psychiatry.

R. de la Fuente: When Dr. López Ibor introduced the concept of "thymopathic equivalents" in his well-known book on "Vital anxiety", some 20 years ago, he rendered us a real service, because he drew our attention to the fundamental, affective nature of a variety of symptoms, both mental and somatic, including some of a seemingly neurological type. His view was that they were related to a basic, vital alteration of mood. The concept of "depressive equivalents" is of course different from, and more specific than, that of masked depression. We must distinguish between cases of, let's say, "camouflaged" depression, and cases in which actual depression is being substituted by one or more organic symptoms. I should now like to ask Dr. López Ibor what are the criteria he uses to establish a diagnosis of depressive equivalents, in the absence of depression. Is it perhaps that equivalents are simply very well-masked symptoms, and that a minor degree of depression is after all present?

J. Angst: The distinction between depressive equivalents and masked depression—i.e. a depression that is present but passes unrecognised—is in my opinion an important one to draw. It makes for a more exact definition of the term "masked depression"—a definition which can be used for operational purposes and enables us to differentiate the condition more clearly from depressive equivalents. Dr. de la Fuente referred to "thymopathic equivalents", a term which I would certainly be in favour of adopting. This more neutral term would be far more comprehensive than "depressive equivalent"; it would avoid the implication that depressed mood must be present and would thus also cover anergic depressive states in which the patient does not feel depressed at all.

H. E. Lehmann: Following the comments of Dr. Angst I would like to suggest that what we should really do is to try to distinguish between thymopathic anxiety equivalents and thymopathic depressive equivalents. The proposal to use the term "thymopathic equivalent" is a very good one, but, from the clinical standpoint and as regards treatment, it would be important to find criteria which would enable us to differentiate between anxiety and depression, as well as between masked depression and masked anxiety.

H. P. Rome: *A propos* of Dr. López Ibor's comments on depressive equivalents, I should like to cite two illustrations. In the paper he presented, one of the neurologists he mentioned was my late colleague Henry Woltman, who reported on what he called the "restless legs" syndrome some years ago. I've had the opportunity to see three of his patients whom he described as being classic examples. In my judgment

these were cases of depressive equivalents and, when they were treated as such, they responded promptly. My second illustration concerns patients with carpal-tunnel syndrome, which presented in classic fashion in conformity with the criteria of SCHULTZE's acroparaesthesia. I was able to follow these patients up after orthopaedic surgery: their symptoms disappeared for several weeks, only to return in the form of classic depressive symptoms.

H. M. VAN PRAAG: In my opinion the fact that a certain somatic symptom responds positively to antidepressants is by no means convincing proof that the symptom is indeed of a depressive nature. I feel we must beware of making exaggerated assumptions about the specificity of the action of antidepressants. All of us here know that most of the antidepressants do, of course, exert a very good action on certain types of depression; but they also have many other kinds of effect—in the direction of sedation, for example. For this reason, I think we have to be cautious in regarding a response to antidepressant drugs as convincing evidence that a particular somatic symptom is in fact of a depressive nature, is indeed a depressive equivalent.

P. BERNER: The connections between a patient's physical state and various psychogenic symptoms, to which Dr. LÓPEZ IBOR has been drawing attention for many years now, strike me as being of fundamental importance, especially with regard to possible "transitions" between psychogenic and endogenous psychoses. What we are dealing with here, however, is two radically different concepts. On the one hand, we are familiar with pathological conditions in which a somatic change is assumed to be the primary disorder; in these cases, the term "endogenous" depression should not be employed, because we do not yet really know anything about their aetiology. It would be better, as Dr. HEIMANN has done, to label them as cases of "psychotic" depression—provided, of course, that we accept KURT SCHNEIDER's definition of "psychosis" as being a mental disorder of somatic origin. On the other hand, there is the concept that an entirely normal substrate—that is, a normal brain—can react to strain by becoming depressed; in these instances, strain is therefore to be regarded as the primary cause. In addition, there are a number of somatic mechanisms which can be triggered off by external experiences and which lead subsequently, via a kind of learning process, to the development of psychoreactive disorders. The "psychotic" disturbances as defined by KURT SCHNEIDER, however, also feature somatic correlatives. The confusion between the two concepts is no doubt due to the fact that a patient, confronted with a change in his physical state of health, may react psychically via mechanisms which we call "psychogenic". Dr. LÓPEZ IBOR rightly emphasised that neurotic depression is in many cases based primarily on physical substrates. FREUD had already pointed out that a third group of factors intervenes between constitutional—or, to be more exact, predisposing—factors and psychic influences. In this connection, it would be interesting today to know to what extent physical predisposition to, for example, psychotic depression—or, more generally, a "thymopathic" predisposition—can influence certain learning processes such as the conditioning, generalisation, and elimination of "neurotic" or also somatic patterns of behaviour.

J. J. LÓPEZ-IBOR ALIÑO: Regarding the problem of distinguishing between anxiety equivalents and depressive equivalents, I should like to mention that, when working on depressive equivalents, I also studied the distribution of the different equivalents by reference to age, and I didn't discover many differences, except in the psychic symptomatology. I found that vital sadness, as defined by KURT SCHNEIDER, played a bigger role in the histories of the older patients, and that vital anxiety, as described by LÓPEZ IBOR, was more prominent in the younger patients. This is illustrated in Figure 1, in which the broken line represents vital sadness, and the continuous line vital anxiety. Here you can see that the continuous line at first falls away with increasing age, but then rises again in the older age groups; this rise is accounted for by so-called involutional depressions, which tend to be associated with anxiety. This figure, how-

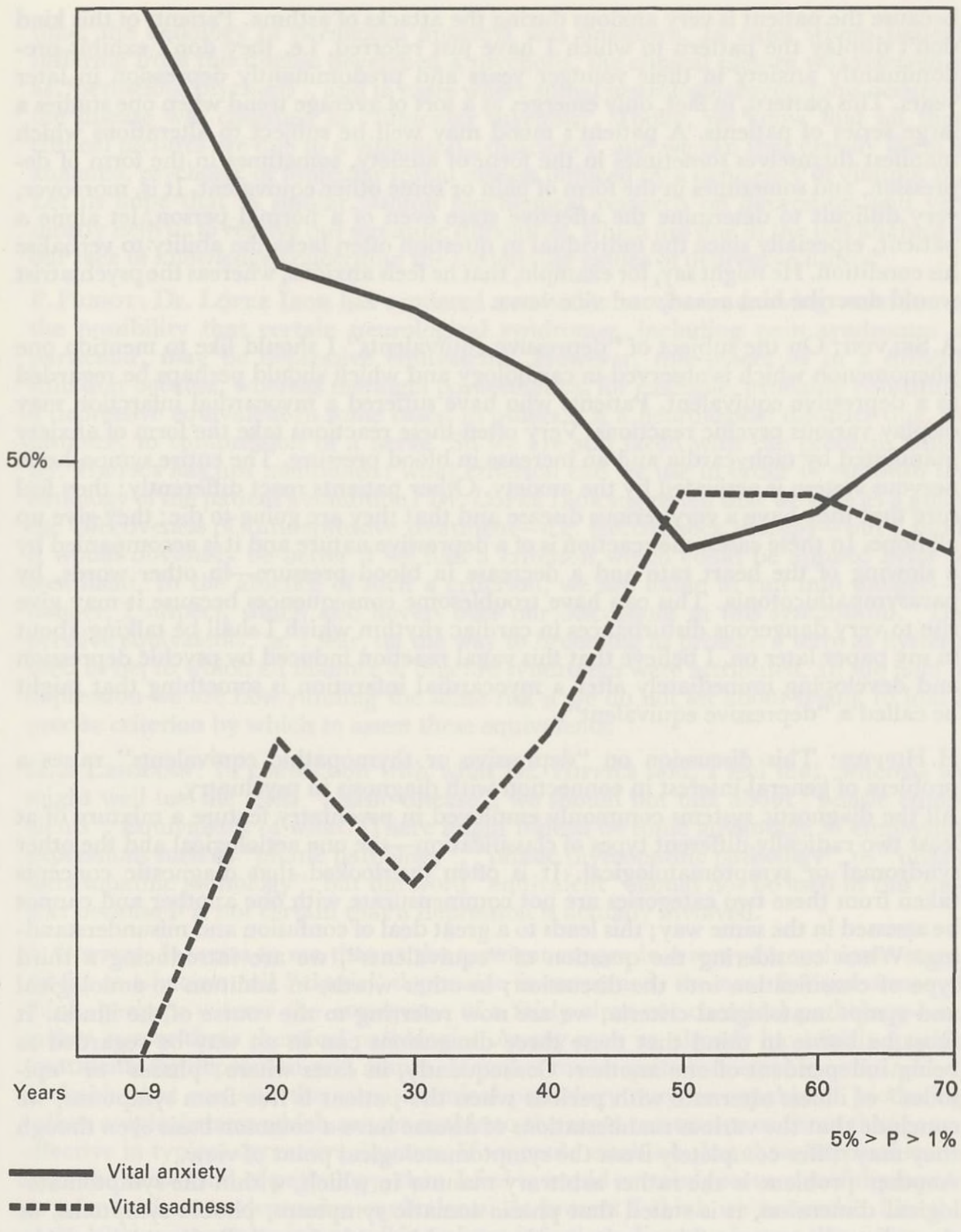

Fig. 1. Incidence of vital anxiety and vital sadness in relation to age.

ever, is based on statistics for over 300 patients*, and not all cases, of course, conform to this age-related pattern. For instance, one quite frequently encounters cases in which the patient has a typical depression—showing a symptomatology characteristic of so-called endogenous depression—and in which this alternates with bronchial asthma; I think the term "anxiety equivalent" might well be applicable to such cases,

* López-Ibor Aliño, J.J.: Los equivalentes depresivos (Paz Montalvo, Madrid 1972)

because the patient is very anxious during the attacks of asthma. Patients of this kind don't display the pattern to which I have just referred, i.e. they don't exhibit predominantly anxiety in their younger years and predominantly depression in later years. This pattern, in fact, only emerges as a sort of average trend when one studies a large series of patients. A patient's mood may well be subject to alterations which manifest themselves sometimes in the form of anxiety, sometimes in the form of depression, and sometimes in the form of pain or some other equivalent. It is, moreover, very difficult to determine the affective state even of a normal person, let alone a patient, especially since the individual in question often lacks the ability to verbalise his condition. He might say, for example, that he feels anxious, whereas the psychiatrist would describe him as sad, and vice versa.

A. Selvini: On the subject of "depressive equivalents" I should like to mention one phenomenon which is observed in cardiology and which should perhaps be regarded as a depressive equivalent. Patients who have suffered a myocardial infarction may display various psychic reactions. Very often these reactions take the form of anxiety manifested by tachycardia and an increase in blood pressure. The entire sympathetic nervous system is activated by the anxiety. Other patients react differently: they feel sure that they have a very serious disease and that they are going to die; they give up all hope. In these cases, the reaction is of a depressive nature and it is accompanied by a slowing of the heart rate and a decrease in blood pressure—in other words, by parasympathicotonia. This can have troublesome consequences because it may give rise to very dangerous disturbances in cardiac rhythm which I shall be talking about in my paper later on. I believe that this vagal reaction induced by psychic depression and developing immediately after a myocardial infarction is something that might be called a "depressive equivalent".

H. Hippius: This discussion on "depressive or thymopathic equivalents" raises a problem of general interest in connection with diagnosis in psychiatry.
All the diagnostic systems commonly employed in psychiatry feature a mixture of at least two radically different types of classification—the one aetiological and the other syndromal or symptomatological. It is often overlooked that diagnostic concepts taken from these two categories are not commensurate with one another and cannot be assessed in the same way; this leads to a great deal of confusion and misunderstanding. When considering the question of "equivalents", we are introducing a third type of classification into the discussion; in other words, in addition to aetiological and symptomatological criteria, we are now referring to the course of the illness. It must be borne in mind that these three dimensions can in no way be regarded as being independent of one another. Consequently, in cases where "phases" or "episodes" of illness alternate with periods when the patient is free from symptoms, we conclude that the various manifestations of disease have a common basis even though they may differ completely from the symptomatological point of view.
Another problem is the rather arbitrary manner in which, within the symptomatological dimension, it is stated that phasic somatic symptoms, phobic symptoms, or sleep disturbances are "depressive or thymopathic equivalents". Perhaps it might be advisable to replace these designations with the more non-committal expression "phasic equivalent", even though this term, too, is not a very happy one. Nevertheless, it has the advantage of being merely descriptive and does not imply that any overhasty hypotheses are being established regarding the aetiology of the various symptomatological manifestations of the disease.
We hope that we shall soon have a well-defined biochemical criterion (e.g. deficiency of free plasma tryptophan) which will enable us to diagnose, and differentiate between, widely varying pathological manifestations. If this hope which we have placed in biochemical research on depression is one day fulfilled, we shall then have trodden the same path as we have already followed, for example, in the case of all those various clinical pictures which are due to vitamin B_{12} deficiency. For a long

time, the aetiology of pernicious anaemia was unknown. In cases where patients suffering from this disease developed a psychosis, the clinical picture was referred to as "pernicious psychosis", and in cases where spinal neurological symptoms occurred, the disease was classified as "funicular myelosis". Today, we know that all these clinical pictures are due to a vitamin B_{12} deficiency, and we can thus classify them as anaemia, psychosis, or funicular spinal disease induced by a deficiency of vitamin B_{12}. Let us hope that in similar fashion biochemical research on depression will very shortly help us to resolve our semantic difficulties, which are due in the last analysis to our lack of knowledge concerning the underlying causes of depressive illness.

P. PICHOT: Dr. LÓPEZ IBOR has rendered a valuable service in drawing attention to the possibility that certain neurological syndromes, including pain syndromes in particular, may be depressive equivalents. However, this places us in a difficult situation because, whereas meralgia paraesthetica, for example, probably constitutes a depressive equivalent in some cases, in others it is a neurological disorder due, for instance, to compression of a nerve. So we are still faced with the problem which Dr. HIPPIUS has just mentioned: what criterion can we use in order to establish the aetiology of the patient's condition? Dr. LÓPEZ IBOR suggested that we take as a guide the effectiveness of antidepressive treatment—e.g. electroshock therapy—but I think it would obviously be better if we had a criterion which could be applied prior to treatment. In the absence of such a criterion, we are liable to run into danger—a danger, I suspect, which also hovers over our discussions at this symposium. "Depressive equivalents" is, I fear, on the way to becoming as protean a term as hysteria used to be, to which all manner of obscure syndromes were attributed. In the field of depression we are now running the same risk if we do not set about trying to find a precise criterion by which to assess these equivalents.

H. E. LEHMANN: In connection with what Dr. HIPPIUS said, I feel that, whereas we might well use the term "phasic disease", we should not talk about "phasic equivalents". Equivalents of what? There might indeed be some advantage in employing expressions such as "phasic pathology", "phasic thymopathic pathology", or "phasic somatopathic pathology", but the word "equivalent" should not be used in this context because it is not certain that a depression is actually involved.

A. COPPEN: It seems to me that at the moment we are in a sort of transitional stage. As far as what we call "classic" depression is concerned, the next few years may, I think, perhaps witness the emergence of a fairly clear-cut chemical pathology—or, rather, two or three chemical pathologies. At any rate, we already have fairly specific treatments for depression and mania. It strikes me as quite conceivable and, indeed, probable that one and the same chemical pathology may be capable of producing rather atypical states which are amenable to the same treatments as those that prove effective in typical depressive illnesses. If we could establish that these atypical states have the same kind of pathology, then I feel it would certainly serve a useful purpose to refer to them as depressive equivalents. At present I think we are rather like the early 19th century physicians talking about infectious diseases; just as they were, we are now faced with a relatively small number of symptoms, and we find it very hard to arrive at any sort of rational classification. But perhaps if we meet again in ten or 15 years' time, we shall be able to discuss this whole problem more profitably, because I hope that by then we may have a sound basis on which to classify these equivalents. We might then be able to take serotonin deficiency, for example, and see precisely what different clinical forms it is liable to assume—apart from that of, say, classic depression.

W. GRÜTER: Dr. LÓPEZ IBOR's reference to the iron deficiency found in cases of "restless legs" puts me in mind of the iron deficiency reported in patients suffering from depression—an iron deficiency, moreover, which quite often proves resistant to treatment. The significance of this deficiency, a problem which has recently been

re-examined by KUHN, as well as by FISCHBACH*, is still rather unclear. I should therefore like to ask Dr. KUHN whether, among the many depressive patients in whom he has carried out serum iron determinations, he has ever come across any cases of "restless legs" associated with iron deficiency. Can any aetio-pathogenetic correlations be established here?

J. J. LÓPEZ-IBOR ALIÑO: I think the example of vitamin B_{12} deficiency which Dr. HIPPIUS quoted is also possibly a dangerous one, because it might tempt us into assuming that underlying all forms of depression there must be some form of deficiency. Let us take tryptophan as an example. When we use tryptophan in the clinic we have to give it in daily doses of 5, 6, or 7 grammes in order to produce a therapeutic effect, whereas when we administer psychopharmaceuticals we can get good responses with only microgramme or milligramme doses. I therefore doubt whether a tryptophan deficiency is the sole factor involved. It is also not merely possible, but highly probable, that the drugs we are using not only exert effects on the central nervous system, but also have peripheral effects—like hydantoin, for instance, which also influences cardiac conduction, or the alpha and beta blockers, which display both central and peripheral effects. Consequently, as Dr. PICHOT has already pointed out, the anti-depressant drugs we prescribe may well be producing an antidepressant action on the central nervous system, plus a peripheral action as well. This might explain, for example, why patients complaining of pains in the legs experience relief, i.e. the drug may be exerting an effect on some biochemical alteration in the peripheral nervous system which is similar to the biochemical changes affecting the central nervous system.
Another point we should not forget is that depressive equivalents were described long before the advent of the antidepressant or thymotropic drugs, and that there have also been reports of cases in which electroshock treatment proved useful in relieving pain associated with psychic illness. KENNEDY**, the great American neurosurgeon, himself applied the term "affective equivalents" to pains in the face or in other areas which responded to electroconvulsive therapy. Though electroshock probably has only a central action, its use may thus nevertheless also be accompanied by peripheral effects—and I think that this point should be borne in mind when considering the therapeutic activity of antidepressant drugs.

A. M. FREEDMAN: May I just add a word of warning with regard to the proliferation of diagnostic categories. I think that, when one presents a new diagnostic category, there is always a danger of "premature closure", a danger of implying that we may know more than we actually do.
We are now in the middle of an important developmental phase with respect to our knowledge of depressions. Within the space of only a few years we have succeeded in producing effective antidepressant drugs, we have achieved real progress in our genetic research on bipolar illness, and we are beginning to dissect out from the group of the depressions various specific categories that are recognisable and whose various manifestations obscuring the fundamental disorder are becoming clearer. It is essential that we should continue this work, in the hope that ultimately we may build up a really sound nosological basis and be able clearly to recognise the various forms which the symptomatology may take. Dr. COPPEN has referred to the problem which the infectious diseases posed for 19th century physicians; it may be that, when we refer to "depressions", we are at just about the same point as they were, a century or more ago, when they spoke of "fevers".
This brings me to the practical question as to the danger of assigning various obscure and complex syndromes to the diagnostic category of depressions. In doing this, are

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* FISCHBACH, R.: Personal communication

** KENNEDY, F.: The neuroses: related to manic-depressive constitution. Med. clin. N. Amer. *28*, 452 (1944)

we perhaps running the risk of foreclosing further study and comprehension of an illness in which depression is secondary? We may aid progress by recognising and accepting present-day ambiguities and uncertainties, because these ambiguities and uncertainties will stimulate further investigation.

A number of the conditions with which we are concerned really involve complex combinations and interactions. If we study cases developmentally, we find, for example, that the child who has asthma from a very early age will react with anxiety and depression not only to the attacks of asthma but also to the grave concern shown by the parents. Some mothers come every half-hour to see if the child is still alive during the night, because they are afraid of a fatal nocturnal attack. This concern communicates something to the child, with the result that, as the years go by, a complex interrelationship develops which cannot be categorised. The disturbance seen later in life cannot be stated to be a concomitant of asthma or a reaction to overanxious parents; it is the result of a concatenation of a host of variables.

H. M. VAN PRAAG: I want to underline Dr. FREEDMAN's statement. I think we should beware of trying to establish facile connections and correlations between certain biochemical findings in depression and certain psychopathological concepts. In experimental psychiatry it is at the moment difficult enough even to get some impression —some quantitative impression—of the amount of anxiety and the amount of depression present in a given case; but I feel sure that it is of the utmost importance to have some impression when evaluating the biochemical and other biological findings in depressive patients. Before we can even think of the biochemical pathology of depressive equivalents or anxiety equivalents, we have to know first of all how to diagnose depressive equivalents, how to diagnose anxiety equivalents, and how to differentiate between anxiety and depressive equivalents. Moreover, if a patient—to revert to one of Dr. HIPPIUS's points—appears to have two or three different phases in his life, we must also ask ourselves: is the disease really a phasic one or are these phases independent of one another? Consequently, before we can tackle the question of biochemical pathology in this very important and very difficult field of depressive and anxiety equivalents, I think we shall have to know much more than we do now.

H. HEIMANN: With regard to the comments of Dr. HIPPIUS and Dr. PICHOT, I should like to point out that we are at present in danger of being swamped by a dynamic phase in medical thinking. There have always been periods when doctors indulged in meticulous descriptions and classifications of syndromes, and there have been periods when everything was once again lumped together under one heading. Dr. PICHOT mentioned hysteria; we could also add neurasthenia. In psychiatry, too, we find both extremely detailed descriptions of psychotic syndromes and, at the same time, the concept of psychosis as a single entity. It's all a question of ebb and flow! As Dr. HIPPIUS said, we are accustomed to think within different frames of reference. But, as long as we are unable to establish strict correlations between the various planes—the phenomenological, the psychophysiological, and the biochemical—which would then make it possible for us to differentiate types that can be defined on all three planes, our discussions are bound to remain far too academic. Hence, I would plead for a certain modesty and intellectual reserve until we know more.

R. DE LA FUENTE: One very typical situation we all encounter in clinical practice is the kind of case in which symptoms completely unrelated to the depression increase and become the patient's central complaint when he is depressed. This sort of clinical situation is quite distinct from other cases where a symptom that is usually present as a component of the depressive syndrome becomes the main complaint. There is still another situation I would like to mention: an habitual symptom from which a patient has been suffering for a long period of time—for instance, headaches—may disappear when he becomes depressed, only to return again when he recovers. This illustrates another aspect of the very complex relationship existing between the somatic and the psychic in the area of depression.

R. KUHN: I was most interested in what Dr. LÓPEZ IBOR said about iron deficiency and its connection with certain motor symptoms. For some time now I have been studying the relationship between iron deficiency and depression by performing serum iron determinations.

In answer to Dr. GRÜTER's question I would say that, as we all know, antidepressants frequently provoke extrapyramidal motor disturbances. Dr. LÓPEZ IBOR also pointed this out briefly in his paper. There is no doubt, in my opinion, that in patients who are prone to display severe extrapyramidal side effects, such as restless legs or other Parkinson-like and dyskinetic functional disorders, these side effects can disappear if steps are taken to correct a concomitant iron deficiency and other deficiencies affecting electrolytes and trace elements. I know of cases in which antidepressants produced such severe extrapyramidal side effects that they almost had to be withdrawn; once the necessary measures had been taken to regulate the electrolyte metabolism, however, the antidepressant medication could be continued without the addition of antiparkinson drugs to the regimen and without provoking side effects.

In this connection, we should also draw a distinction between organic and functional disorders—for example, between extrapyramidal symptoms and depressive mood. Depression frequently influences the physical symptomatology and may even modify it to such an extent that certain symptoms appear to be of a depressive and exclusively non-organic nature. This also applies to motor symptoms caused by the disease itself or by the medication. Among these motor symptoms are to be found all manner of extrapyramidal disorders, including restless legs. Hence, an organic disease—for instance, of the locomotor apparatus—may give rise to much more marked signs and symptoms in a depressive patient than in a mentally normal subject. *This, however, does not mean that the symptoms are of a psychic nature.*

J. J. LÓPEZ IBOR: It would take up too much time if I were to comment at length on every point that has been raised, and I therefore consider it preferable simply to make a general summary of this discussion.

First of all, I would point out that the term "masked depression" is being used in the United States even in books intended for general practitioners. According to my information, one of the first people to employ it was the famous neurologist FOERSTER KENNEDY shortly after the Second World War.

Generally speaking, in Central Europe the term "*larvierte Depression*" is more commonly employed. Apart from these expressions, in 1950—in a book published in Spanish, in which I reviewed articles that had appeared some years previously—I spoke of *thymopathic equivalents*. In this book I expounded the problem of the existence of a neurotic anxiety that was qualitatively different from either normal or reactive anxiety.

Reactive feelings are those that are produced by events in the external environment. It is strange that in the study by SCHELER sadness is listed only among the reactive feelings and not among the vital feelings. Consequently, in an attempt to suggest the existence of a "thymopsyche"—and thus to make a tripartite description of the personality instead of the bipartite Cartesian description—I used the expression *thymopathic anxiety* for a group of neuroses whose psychodynamics did not appear to explain either their presence or their symptomatology.

FREUD, when discussing the question of anxiety, spoke of *anxiety equivalents*, and in his publications we find a description of these equivalents which amounts to nothing more than an enumeration of the vegetative symptoms that often accompany crises of anxiety.

Since I considered that, as FREUD himself says, anxiety was the "core" symptom of neuroses, I proceeded to extend the category of the neuroses by including in it anxiety of the type that I then called *thymopathic* in order to distinguish it from the other types.

My own clinical experience, supported by the findings and opinions of a number of other psychiatrists, convinced me that, despite the views of KURT SCHNEIDER, there

was an imperceptible transition between endothymic anxiety and the sadness that we could also call endothymic. The person suffering from melancholia or from depression is a person who is unable to be sad (SCHULTE)*.

A detailed analysis of the life experiences reported by patients—both by those suffering from neurosis or depression and by others who came to my consulting-room with different diagnoses, some of them neurological—compelled me to state the problem to myself in the following form:

Among the depressions there are some which pass without being perceived, and these are unknown or masked depressions. The case that I later depicted in a film entitled "Antonia L."** demonstrated to me the frequency of these depressions, and, as the years went by, I encountered a steadily increasing number of such cases. However, since I dealt with this point at last year's symposium***, I shall not dwell on it any further here.

FREUD's description of the anxiety equivalents raised for me the problem of the equivalents met with in some patients who at times are diagnosed as suffering from atypical depression and at other times even as suffering from neurosis. My attention was first drawn to this problem by a patient who presented initially with perineal pain referable to a previous gonococcal infection and who, after having consulted leading urologists and other specialists in various parts of Europe and the United States, was sent to FOERSTER KENNEDY. To cut a long story short, FOERSTER KENNEDY treated him with electroshocks****. Subsequently, the patient had phases, with long intervals between them, either of pure depression or of depressive equivalents.

Since then, we have studied the question of whether many of the atypical pictures observed in patients who go from clinic to clinic without obtaining a more or less clear diagnosis might not be depressive or affective equivalents. Dr. LÓPEZ-IBOR ALIÑO***** has written a very detailed thesis on the cases of this type which we have encountered in recent years.

Hence, besides depressions that can be called masked or *larviert* or by some other similar term, there also exist forms which might be referred to as incomplete, because all that the patient complains of is a symptom, and virtually nothing he says is directly suggestive of an underlying depression. A description of some of these symptoms is more likely to be found in the works of the classic neurologists than in those of classic psychiatrists, with the exception of GRIESINGER.

English authors prefer to speak of affective equivalents rather than depressive or anxiety equivalents. What is important, however, is that the whole question opens up a new line of investigation.

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* SCHULTE, W.: Studien zur heutigen Psychotherapie (Quelle & Meyer, Heidelberg 1964)

** "Documenta Geigy" Films

*** LÓPEZ IBOR, J.J.: Masked depression and depressive equivalents. In Kielholz, P. (Editor): Depressive illness, Int. Symp., St. Moritz 1972, p. 38 (Huber, Berne/Stuttgart/Vienna 1972)

**** KENNEDY, F.: Personal communication

***** J.J. LÓPEZ-IBOR ALIÑO: Los equivalentes depresivos (Paz Montalvo, Madrid 1972)