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CLINICAL MANIFESTATIONS OF AUTISM AND SCHIZOPHRENIA IN CHILDHOOD

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THOUGH PSYCHOTIC MANIFESTATIONS occur in childhood with mathematical regularity – in 1 out of every 200 children–general practitioners and pædiatricians do not appreciate them sufficiently.

In 1896 Kraepelin distinguished dementia præcox from "insanities", thus emphasizing its appearance in early life. He stated that 3.5% of what we now call the schizophrenias begin in childhood. In 1943 Kanner described the syndrome of early infantile autism on the strength of his collection of 11 such cases; by 1949 he had recognized this condition in 750 children. In a recent clinical study Bender (1947) reviewed 100 children with schizophrenia seen in the past 10 years at the Bellevue Hospital, New York.

This paper is offered to the general medical reader because of the paramount importance of early diagnosis in the prevention and treatment of schizophrenic disorders in childhood. The clinical material studied includes 5 definite cases and several less definite cases in which diagnoses varying from schizoid personality to autism and schizophrenia have been made. These children have been studied in a child guidance setting for periods of over one year each. In addition this writer has gained relevant experience from the analytical study (over a two year period) of one definite case of an adult of 21 who had a schizophrenic disturbance in childhood.

The literature on the subject was found to be profuse but at times confusing. Fifty of the more recent monographs, texts and articles have been reviewed. Authors are handicapped in that the etiology of schizophrenia remains unknown. Thus recognition and classification is based on the description of clinical manifestations. But the behaviour, thought and feeling of a person disturbed psychiatrically is as varied and variable as the total life setting, and the personal experience of the individual. The difficulty is greatly augmented when children, about whose mental processes we still know so little and who change and grow so rapidly (in comparison with adults), fall ill with psychiatric complaints. What seems to be needed to make description more meaningful is a uniform systematization of the material studied and an adequate formulation of the manifestations encountered. In the present paper efforts are made in this direction by hypothesizing a range of clinical manifestations of the schizoid kind occurring in the life period from birth to adolescence.

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CLINICAL MANIFESTATIONS

The nosological problem hindering our progress in understanding could be helped by realizing that most of the symptoms and signs in all but the most disturbed children may be seen at times in the average child. What renders them pathological is (a) The special constellation of symptoms. (b) The persistence or progressive deterioration in the disturbance. (c) The appearance of persistent behaviour not in accordance with chronological age; that is, fixation or actual regression.

Perhaps the most striking exceptions to this are the lack of spontaneity in the infant who refused to be cuddled and who will not put out his arms to be picked up, seen in infantile autism, and the peculiar postural reflex responses of the schizophrenic child, described by Bender. Yet even these only become pathognomonic when regarded in conjunction with the rest of the clinical picture.

The writer sees the manifestation of these disorders in a broad spectrum in which at one end there is the average child, and at the other end there is the grossly disturbed child. Somewhere along the line there must be a considerable accumulation of etiological factors, psychogenic and organic, to make for irreversible pathology. For the sake of clarity five arbitrarily delineated groups of manifestation will be considered. The terms categorizing them are admittedly limp, yet one is unwilling to coin more jargon.

1. *Neurotic manifestations.*—The child's fundamental personality feature is shyness with fearfulness. The reactions ensuing may be withdrawal and introversion, thus resulting in impeded development or even regression. The central paranoid mental structure found by Klein to be common to neurotic children, may be much in evidence. The development of further ego-defensive reactions, such as phobias and obsessions, is of malignant import. They may range from fear of fantastic events occurring in the dark, to rituals involving much of the psychic

scene. At one critical point both the timidity-withdrawal affective component and the phobic-obsessive conative component may involve the total personality. Then the resultant morbidity would be too great to include such a case in this near normal group. At that point the child will seek seclusion and his thinking may fail to integrate parts with wholes. Such a child may even forsake his mother's comforting care. He may fear such naturally occurring events as falling leaves and be upset by such incongruous details as having to recite the names of all edible vegetables before eating. Yet there are many children, especially of pre-school age, who exhibit some such features, and some who may show one or more of the following behaviour symptoms, which Bradley and Bowen (1941) and others consider significant for schizophrenia.

(1) Seclusiveness. (2) Irritability when activity is disturbed (Both these are regarded by Bleuler as primary symptoms of schizophrenia). (3) Day-dreaming. (4) Bizarre behaviour and toilet habits. (5) Diminution and regression of interest). (6) Sensitivity to criticism. (7) Physical inactivity and compulsive phenomena. To this list may be added some of Potter's (1933) criteria for diagnosing schizophrenia. (8) Alteration and fluctuations of behaviour with increased or decreased psychomotor activity. (9) Defect in emotional rapport.

If, in the history, there is severe emotional trauma, physical neglect or hurt, psychologic rejection, shock or deprivation—in one word, if the child had been "burnt"—then many of these manifestations can be ascribed to a neurotic reaction and are likely to be modified and even resolved. The onset is likely to be acute and sometimes it may resemble the catastrophic reaction.

2. *Schizoid psychopathic personality.*—In the absence of a history providing the factor of acute severe external stress, the structure of personality is likely to be faulty, either because of protracted and insiduous external stress (such as a cold, obsessive personality of both parents) or because of an inherent flaw. In this case also the onset may be acute or even catastrophic and precipitated by minor physical or emotional upset. More usually it is insidious and the condition involves the personality to a greater extent than in an neurotic reaction. The cardinal manifestations are withdrawal, some fragmentation of the psychic process and loss of spontaneity. This child is cold and unapproachable. He may whine and cry incessantly, be restless or grossly inactive and indulge in much day-dreaming. Many of the characteristics of autism may be included: immature speech, inability to abstract from experience, a compulsion for sameness and a low threshold for tolerating interference with his activities. In the absence of a history of autism dating back to the first two years of life, and provided not all areas of adaptive functioning are involved, so that there is sufficient integration for average or below average progress in the home and school, such a patient can be categorized in the group of psychopathic personality without yet falling into the syndrome of autism. The incidence of mental illness in the family is not necessarily high. The child may show obvious evidence of physical immaturity in physiognomy, E.E.G. pattern, physiologic responses, etc.

There is an implication in these two groups (neurotic and psychopathic) that psychopathologic factors play the major rôle in etiology and constitutional factors are of secondary importance.

3. *Early infantile autism.* — Though some workers doubt the existence of this syndrome as an entity, autism is a manifestation seen throughout the schizoid range of disorders, and it forms a nidus for the schizophrenic process in the child and in the adult. Its basic characteristic is the child's inability to relate to people and situations from the earliest months of life, and this becomes established at the preverbal level of the child's development. Some (Mahler *et al.* 1949) would argue that autism arises secondarily in an attempt at integration; that there is an early defect in ego development occurring at the crucial periods of life, *i.e.,* in the first year and then in the 2 to 5 year period. This results in a libidinal (id-derived) surge threatening the ego structure. In a desperate attempt to maintain a hold on reality the child relates to its less threatening aspects: objects and parts, rather than to its more dangerous aspects: persons and wholes. Then the mental function of symbolization fails to develop. This argument begs the question as to what leads to the original defects and what is its nature. This writer would like to postulate that, as we move in our spectrum of schizoid phenomena towards the more virulent and morbid extreme, autism increases and that at one point of loading it becomes the primary defect. The writer would place the pathological

Canad. M. A. J.
July 1953, vol. 69

locus of this manifestation at the level of the mysterious translation of "organic" perceptions (the result of "mechanical" stimuli from the inner and outer physical worlds) into meaningful and integrated psychic experiences. The vehicle which seems to facilitate this transition between the objective and subjective world and back into the outside world (rapport) is feeling tone—a primary affective association with all perception. It seems to me that this vital association of a feeling in tune with perception is grossly defective in autism. It is particularly blocked or deficient in that area of man-to-man relation which is perhaps the greatest integrating force of the psychic system. Consequently special relations and body image will be disturbed *pari passu*. The locus of the defect is placed at the fundamental psyche-soma linkage. If the weight of etiological factors is psychic, the condition may be reparable to an extent, if somatic probably less so.

In summary, autism is a fundamental *disarticulation* between the child and his world. This formulation helps to put into perspective its clinical manifestations, which are recorded below in the systematic order in which it is suggested that such data be uniformly recorded in case histories.

Behaviour manifestations:

1. Interpersonal (social) relation: little or no rapport; irritable if day-dreaming or compulsive behaviour is interrupted.

2. Relation to objects: good with some emotional rapport; relatively free projection of phantasy and emotions on objects. A desperate need to keep *sameness* in arrangements of toys, furniture, play patterns, etc. Absorbed in details, apparently ignoring the total object, *e.g.*, a child may be angry with the foot which trod on one of his pencils, yet ignore the owner of the foot.

3. Relation to self: difficulty in fusing the idea of self to *himself* and frequent misuse of the pronoun "I"; consequent distortion of image of his physical self.

4. General psychomotor behaviour: level of consciousness maintained throughout awake-period; often hyperkinetic but also dreamy and detached; usually plays in a co-ordinated and prolonged fashion with play things.

5. Mood: impassive but sometimes extremely fearful; low threshold for frustration tolerance, cold and withdrawing.

6. Conation: dereistic thinking; phantasies often of great danger. There is a literalness and concreteness to all thinking. There is lack of appreciation of total meaning.

7. Speech: echolalia; idioglossia; speech is not used primarily for communication; it is mechanical and affectless; literalness involves most words and prepositions; there may be mutism.

8. Compulsive phenomena: marked repetition compulsion of words and activity; consequently excellent rote memory.

9. Cognition: perceptions are clear and potentially good. There may be an apparently paradoxical phenomenon in that the sense of actual physical pain may have a high threshold and not be easily aroused, while the psychic threat of minor physical pain may cause a high degree of emotional disturbance: fear.

10. Intelligence: defective in total function and especially in inductive reasoning; yet preoccupation with abstract subjects; failure to symbolize. Impossible to gauge potential since communication is impaired.

In the history, the time and the type of onset has already been noted. The family history may be remarkable for obsessive trends and particularly notable for the high incidence of perfectionistic parents and relatives, who have achieved good professional status owing to their high mechanically intellectual ability. They very frequently mistake their ill offspring for a genius because of his extraordinary exhibition of rote memory and his serious, preoccupied and withdrawing nature.

4. *Schizophrenia.*—The difficulty in the formulation of a frank schizophrenic reaction in childhood is that its main characteristic is resemblance to the adult reaction. This includes the "silliness" of hebephrenia, the odd postures of catatonia and the persecutory and grandiose delusions of the paranoid type. Now, for such definite patterns of reactions to arise, one must postulate an important early period of maturation, basically not interfered with by the onset of the schizophrenic process until some time past the main corner-stones of early childhood. Yet this disturbance is the most virulent and disrupting of its kind and involves every level of adaptive functioning. Bender defines it as "a form of encephalopathy" and the constitutional element with its "organic" factor seems heavily weighted. Though Bradley insists that it presents as a true psychotic reaction with a history of difficulties since the earliest years of life, one must agree with the suggestion of others that one cannot expect the schizophrenic child to show the details of reaction of the adult. I would go further to state that if the process started in infancy and continued to involve all adaptive processes, the resultant effect would be chaotic and incompatible with psychosomatic developments. Indeed, such may well be the case in a few early and acute reactions which go on to "deterioration" or rather arrested development of the infantile personality. The writer would suggest, however, that the majority of cases either develop subsequent to a mild phase of autism or insidiously, in the absence of infantile autism, over the first 10 years of life, thus allowing for

a differential maturation of the psychosomatic organism.

The behaviour symptoms are as follows:

1. Social relation: lack of inadequacy or socio-affective rapport; strong ambivalence; secondary symptoms: withdrawal and regressive behaviour, including soiling, etc.; tertiary symptoms: an attempt to cling to people, neurotic trends, severe and persistent temper tantrums, hostile and impulsive actions, like incendiarism and attempted suicide.
2. Relation to objects: may be the same as in autism.
3. Relation to self: distortion of body image, fragmentation of already immature picture of self and distortion of image of others; hypochondriasis; perception of self as isolated.
4. General psychomotor behaviour: (a) Disturbance of vegetative system: vasomotor, sleeping, eating, elimination, abnormalities. (b) Motor: persistence of primitive reflex pattern activity; rotating and whirling, motor play; patterned grimacing and oral mannerisms; odd voice; general hyper or hypo fluctuating activity. (c) Abnormal E.E.G.
5. Mood: Pathognomonic incongruity of affect; loss of spontaneity and immediacy in childish joy and excitement; "brittle" response; anxiety—severe and unaccounted for by the facts of reality; part of this anxiety may be secondary to the psychopathologic process of dissociation and disintegration; there may be alternation of abnormal moods: rage, laughter, crying. The disarticulation with the environment owing to a discordant feeling tone is much in evidence.
6. Conation: failure in the identification process; the ego (the "I") is distorted; loss of plasticity, preoccupation with abstracts; inability to conceptualize; failure to perceive the relation of parts to whole. Essentially, thinking is dereistic and may be delusional.
7. Speech: As in autism, speech is immature and if the process is established before speech then complete mutism results (in about 1/3 of cases). The speech is a fine index of the progress of pathology in the thought process. It may be precocious with an extensive vocabulary at first, and then become increasingly autistic, eventually with bizarre irrelevance, repetition, stereotypy, echolalia, condensation, neologism, pronoun fixation.
8. Compulsive phenomena: May be present as tertiary symptoms but not so pronounced as in autism, for there is a greater failure of ego-defence.
9. Cognition: Unlike in autism this is always somewhat and may be grossly disturbed. There may be grotesque distortion of both persons and objects; fluid ego boundaries; uncertain perception of centre of gravity with whirling motion; there may be florid hallucinations. The incongruity and dissociation between not seeming to feel actual painful stimuli (like a cut or bruise) and yet being hypochondriacal about potential and minor physical or psychic threats (like having a stethoscope applied to the chest or having the nails cut) may be much in evidence.
10. Intelligence: Originally I.Q. function may be high but eventually it will drop with a wide scatter.

The history may show psychopathic loading in the family. The onset may be acute or insidious and, as mentioned, only occasionally setting in at the preverbal level.

Investigations:

1. Psychological tests: The Goodenough draw-a-person, the Rorschach, the Thematic Apperception Test, Vigotzki tests confirm disturbance of body image of temporal and spacial relations, and demonstrate the thinking and emotive defects.
2. Physiological tests: Responses to such pharmacological drugs as histamine and mecholil, E.E.G. patterns, blood-sugar estimations on stress, etc., show the essential immaturity of reaction.
3. Physiognomy and body development also show characteristics of immaturity.

Differentiation from primary mental defects: (Oligophrenia). (1) Early period of normality, in some schizophrenics. (2) Some precocious skill or knowledge, in some schizophrenics. (3) Clinical evidence of the psychotic reaction-type. (4) Psychological tests confirming diagnosis. (5) Observed behaviour indicating high potential intelligence; choice of words beyond age; good memory. (6) Response to therapy.

5. *Dementia præcocissima.*—This is a heterogeneous collection of syndromes with major organic deficit. It includes Heller's dementia infantilis, Tay-Sachs' disease, the encephalitides, various aphasias and agnosis due to differential brain atrophy or abiogenesis. The organic element is detectable and the total personality, including intelligence, is defective. Unlike in the more functional disorders, the child succumbs early to the underlying organic disease.

Course and prognosis.—A child finding himself in one of the broad categories described may pursue a variable course; the shy and fearful child may and usually does become healthy or at worse neurotic; or his fears may augment and diffuse, and timidity become so morbid as to lay down a schizoid structure in the personality; finally he may become schizophrenic (he cannot *become* autistic).

The schizoid psychopath may and usually does retain morbid shyness and an isolated personality with a quality of an arrested schizophrenic process, or he may be swallowed up entirely in a psychotic state.

The autistic child is unlikely ever to be average. Reparative processes may mend and patch the defect to the extent of allowing some functioning of the individual within the limits of a stunted personality.

The schizophrenic child has a poor prognosis. He has a long and protracted fight on his hands, but depending on what may be preserved in the personality and, therefore, partly depending on the age of onset, the child is by no means condemned to a life-long psychosis. In my experience of one such case with adequate help, a fair resolution of symptoms is possible, perhaps within the limitation of a rigid obsessional personality. Broadly, the factors of bad omen are: the setting in of autism in the early months of life; a severe speech disturbance, particularly mutism, which impairs grossly communication with the outer world (and, therefore, a psychotherapeutic approach) and a numerical I.Q. definitely below

70 points. Prognosis depends on early detection and treatment.

CASE MATERIAL

CASE 1 (Autism).

R.B. A boy, aged 5, presented with perpetual fears of objects and animals, he was terrified of puppets, flying paper, dogs and of other children, and also of any unanticipated movement or noise. He was timid, claustrophobic, talked to himself in single, unintelligible monosyllables, and would seek seclusion if hurt, and then perhaps cry all night. His sleeping, eating and habits of elimination were poor. He would habitually bang his head, masturbate, and show rituals in his behaviour when going to bed, eating, etc. His speech was infrequent, autistic, repetitive and echolalic. He would imitate and repeat unexpected sounds until his fears were pacified. He formed clinging relations to some objects and would become hostile if interrupted in his magic and repetitive play activity, which was designed to overcome his fears. He would appear to ignore the presence of adults, but give one the feeling that he felt they were there. As a baby he was said to always have been odd and lacked spontaneity and a show of affection. He has always been extremely sensitive to the slightest unusual noise and to any change in the *sameness* of his environment. The parents were rather cold and shy, and the father had a temper which he over-controlled. There was no overt mental illness in the family. Physically the boy had stigmata of immaturity and was malnourished. On psychological tests his I.Q. was 73, but with a wide scatter. In the playroom he gave the impression of a much higher I.Q. potential.

CASE 2 (Schizoid personality developing schizophrenia).

E.W. A boy of 10, first presented when aged 8 because he did not fit into the school program and had to repeat his grade. He had fluctuating moods with impulsivity, restlessness, noisy periods, ate little, paid attention to minute details and had good motor ability (he learned to ride a bicycle in one day). He was helped considerably by play therapy, forming a fair relationship with the therapist. However, 2 years later his moods led to unmanageable, hostile behaviour. Yet most of the time he seemed dazed and day-dreaming, spoke infrequently and rambled often. He was full of odd gestures and oral mannerisms. The onset was vague and insidious —"he has always been an odd child". The family history was clear, though the parents were markedly schizoid. Physically he was healthy, but an immature looking boy. E.E.G. normal. His I.Q. was 72, but his verbal ability was high, and often he gave the impression of good intelligence even at school.

CASE 3 (Simple schizophrenia).

L.B. A boy, aged 11, presented with day-dreaming, dereistic thinking, incongruity of affect, morbid shyness, enuresis, poor sleeping and eating. He lived almost entirely in a world of his own and appeared vacant much of the time. His I.Q. was 80. E.E.G. normal. There was no history of stress in the environment. Family history showed some psychopathic loading.

CASE 4 (Schizoid psychopathic personality).

R.P. A boy, aged 9, presented with disordered behaviour such as stealing, masturbating, disobedience, noisiness. He had a good rote memory, but poor understanding of words. He had many somatic complaints and many fears. Though he was quite asocial he was none the less capable of forming an emotional rapport when the circumstances were favourable (in the playroom). His I.Q. was 83, yet he gave a strong impression of a potential well-above this level. At his birth his father was already middle age and there was some emotional deprivation, as well as a mild birth injury, in the history.

CASE 5 (Neurotic manifestations).

B.McF. A boy, aged 10, had a severely handicapping congenital heart disease for which he had been operated upon. His world was half of phantasy and half of reality. He grimaced foolishly and acted in a "silly" way, and sometimes incongruously. He tended to withdraw and to become suspicious and resentful when pressed. His mother was obsessional and rejecting. His I.Q. functioned at the normal level (93). He had a remarkable awareness of his own "silliness" and attempted to correct it and bring himself into line with his world.

It will be noted that all these patients were boys. The literature seems to give also an impression of a preponderance of boys in the case material. It will also be noted that I.Q.'s were below average, yet it was felt that the actual test could not reflect the potentially average intelligence.

Etiology.—The etiology of schizophrenia in childhood is unknown. Immaturity of tissue and psychic structure is found throughout this range of disturbance, but it cannot be considered in a strictly causal relationship, because such a disrupting and malignant process as schizophrenia cannot fail to coincide with arrested psychosocial, psychosexual and psychosomatic maturation. Consequently the relationship between at least one significant factor, namely immaturity and the ongoing process of schizophrenia, is a circular one.

Organic and constitutional factors weigh heavily at the extreme end of the spectrum of schizoid manifestations. The most profitable search seems to be for the interplay of a constellation of specific psychodynamic factors with the possible background of an inherent defect-autism.

Treatment.—Physical methods of treatment are generally disappointing. Benzedrine in doses of 5 mgm. several times a day may be helpful, especially where there are E.E.G. irregularities. Electroshock should be used with great discrimination and sparingly, since it is not of established value and is likely to be damaging when applied to a child. Psycho-surgery (lobotomy) is mutilating and should never be considered in a child.

The essence of successful treatment is the early recognition of such morbid prodromal symptoms as excessive shyness, withdrawal and excessive fears, and a referral to a consultant or to a child guidance clinic, where the condition can be properly diagnosed. The most promising method of treatment is long term play therapy and analytically oriented psychotherapy (in later childhood and early adolescence). In this series of cases they have all been improved consider-

ably by play therapy and the suitable alteration of the environment, that is, by modification of parental and school attitude.

CONCLUSIONS

Schizophrenic reactions occur in children, as well as in adults. They tend to produce a picture frequently confused with mental deficiency. They occur more commonly than has been realized. They can be best treated by psychotherapy and environmental manipulation, when it appears that as high a rate of improvement as 20% (Lourie 1943) is possible.

I am indebted to my clinic colleagues, Dr. M. A. Menzies, for his review of the literature; to Miss E. Gardiner and Miss F. McDonald, psychologists, and Miss M. Tucker, social worker, who helped me in the clinical work; and to Mrs. C. Reid, secretary, who helped me with this paper.

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MUMPS MENINGOENCEPHALITIS

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BETWEEN THE MONTHS of December, 1951 and June, 1952 an epidemic of mumps in the rural area of Ponteix, Saskatchewan, afforded us an excellent opportunity to study this disease. We estimate that in a population of 3,500, approximately 250 cases of mumps occurred during this outbreak, the first in this locality since 1937.

Under a regional prepaid medical care program, there was no economic barrier to medical attention; however, severe weather conditions restricted travel and prohibited observation of many cases. While we cannot claim to have seen all the cases, it is probable that most of those following an atypical pattern were reported to us. During this epidemic we established a diagnosis of manifest mumps meningoencephalitis in 39 cases. Descriptions of the illness by telephone during the acute phase, and in personal interviews after recovery, convinced us that many additional cases of mumps meningoencephalitis (MME) occurred.

Despite the growing volume of literature devoted to MME, many fundamental questions regarding its pathogenesis, incidence, diagnosis, and treatment remain unanswered. In this article we shall review the present knowledge of these aspects of MME, and report on our experience with the 39 cases which we were able to follow closely.

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Pathogenesis.—In the past, MME has been attributed to a multitude of causes. Early experiments indicated that the etiological agent was transmissible. Recently, the mumps virus has been isolated from the spinal fluid of patients with MME. It is not known whether the virus or its toxins are responsible for this syndrome.

The path of invasion of the mumps virus to the central nervous system has not yet been elucidated. Philibert advanced the theory that it first attacks the central nervous system, perhaps with the cornea as portal of entry. Transient corneal opacity has been reported in rare cases of mumps. Kilham's failure to isolate the virus from the spinal fluid of patients with *early* mumps parotitis without evidence of meningeal involvement refutes this theory.

It has been shown that the buccal cavity can be the portal of entry for the virus. The long incubation period of mumps supports the belief that the parotitis is not caused by direct extension of the virus along Stenson's ducts, nor the meningoencephalitis by invasion through the roof of the nasopharynx. The same evidence has suggested the theory that the mumps virus, like that of rabies, reaches the central nervous system via the peripheral nerves. The wide dissemination of mumps infection through the body is the basis for the currently popular theory that mumps is a viremia with a predilection for nervous and glandular tissues.

Diagnostic criteria.—The absolute diagnosis of MME requires, as does the diagnosis of pulmonary tuberculosis, the isolation of the etiological organism from the affected tissues or their secretions. Any less-exacting criteria leave room