Systems & Symbiosis - The Bowel Nosodes Reappraised

v5.0 January 2007

A Seminar in Integrative Medicine

Core Text

Course Text & Study Resources

by

Russell Malcolm FFHom.

.... if the cause maintaining the disease still persists. Some circumstance is to be found in the regimen or the environment of the patient which must be eradicated if the cure is to permanently come to pass.

Samuel Hahnemann *Organon of the Medical Art*
Para. 252 Trans. Steven Decker

Preface

There are few things more rewarding than seeing someone recover from longstanding illness: to regain their lost energy, creativity and sense of belonging. However, in previous years, a certain proportion of people in my care showed disappointing responses to carefully worked out treatments. This was disheartening for a physician with years of study and thousands of 'patient-hours' behind him.

The introduction of a group of immunotherapeutic medicines, known as the 'Bowel Nosodes', has transformed outcomes in many of my chronically ill patients. It is my hope that others involved in chronic disease management will observe similar 'quantum leaps' in their 'stuck' patients, as they incorporate these materials into their therapeutic armamentarium..

Any clinical discussion dealing with the microecology of the bowel would not be complete without a dedication to Drs. Edward Bach and John Paterson. Their exhaustive bacteriological investigations and clinical correllations spanned almost 40 years. They systematically gathered data from many hundreds of patients and analysed thousands of bacteriological cultures.

It is unfortunate that their contribution to the fledgeling science of clinical bacteriology has been so seriously overlooked by the medical community. This historical injustice is partly due to the paradigm within which they worked. Even now, the subject of medical homeopathy can still evoke a knee-jerk rejection in some medical minds, regardless of the rigour and quality of the scientific enquiry.

Collective clinical experience is difficult to ignore, however. Particularly when it involves clinical responses in chronic systemic illness; in patients who have been increasingly refractory to standard drug treatment. When the case literature is taken together with recent scientific advances in intestinal microecology, it becomes clear that a reappraisal of Bach's and Paterson's work is long overdue.

This book has been written primarily for doctors with homeopathic training and experience. It is hoped that general physicians, gastroenterologists, bacteriologists, immunologists, medical historians, and those involved in the treatment of chronic systemic illness, will find the hypotheses and therapeutic ideas stimulating and clinically helpful.

The experience of my patients has been the primary motivation for writing, and I am grateful to those of my patients who have allowed me to use their case in the clinical sections.

Russell Malcolm - summer 2002

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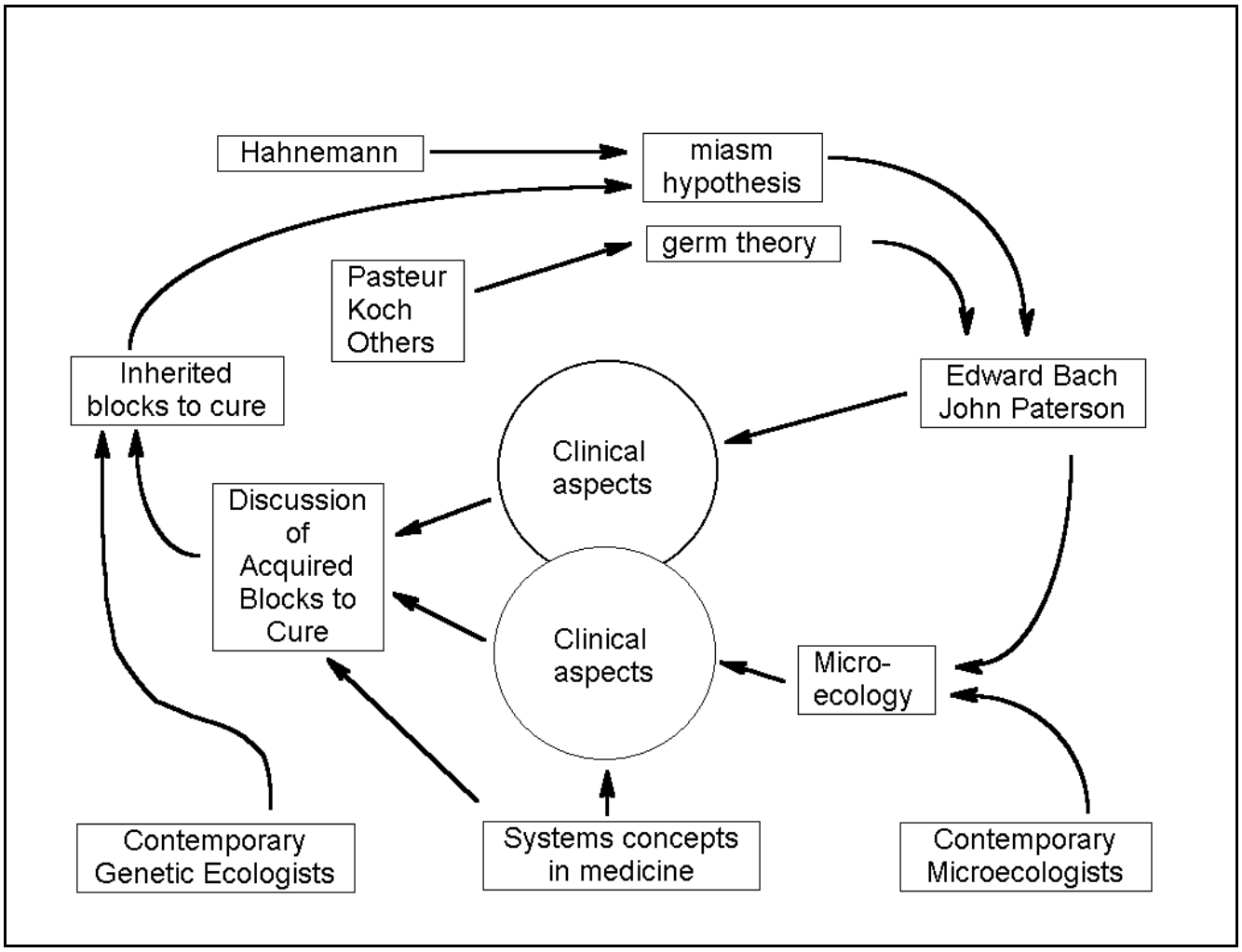
The Bowel Nosodes Reappraised

Aims and Objectives of the Seminar:

The main aim of our weekend together will be to deepen our knowledge of the bowel nosodes through collective discussion of our clinical experiences and a structured programme of study.

To make the best use of our time we will use a framework which progressively integrates orthodox and homeopathic theories of acquired illness - and acquired blocks to cure.

We will begin with a brief review of Hahnemann's miasm theory and end with a re-examination of the same ideas.



Objectives: By the end of the weekend you will:

- have some knowledge of the history and origin of the bowel nosodes
- understand the concept base that has governed their use
- have an awareness of systems concepts of illness and how these relate to the nosodes
- have a clear knowledge of the main clinical indicators for their use
- be familiar with the possible methodologies for their incorporation into the case strategy
- understand the basis of their clinical relationships to classical homeopathic remedies
- have an awareness of their potential as a focus for improving integration within medicine

Introduction

The treatment of chronic disease is 'stock and trade' for medical homeopaths. The successful treatment of chronic illness can be immensely rewarding for patient and healer alike. However, the treatment of systemic illnesses often presents frustrations and challenges, particularly when our patient is trapped in a precarious relationship with various drug therapies, or where there are other significant blocks to cure.

One significant 'block to cure' is intestinal dysbiosis. Apart from undermining the patient's ability to respond appropriately to homeopathic treatment, intestinal dysbiosis can generate complex patterns of systemic illness. If dysbiosis is not recognised, the patient will respond poorly to what are ostensibly well chosen treatments.
When such well individualised treatment fails, opportunities for cure are often missed as patients commit themselves to long-term symptomatic drug treatment.

The primary objective of this seminar is to help avoid such missed opportunities, firstly, by helping practitioners to recognise the main features of intestinal dysbiosis and, secondly, by providing some practical guidance to its eradication.
We will first describe the most frequent clinical manifestations of intestinal dysbiosis. We will then discuss how it can be treated using bowel nosodes, either on their own or with their related homeopathic medicines. In the clinical sections we will outline the main indications for incorporating bowel nosodes into a wider treatment plan.

In 1988 the British Homeopathic Journal published a series of papers on the bowel nosodes [59] [60] [61] [62] [63] [64] [65] [66] [67] . These volumes represent the most coherent survey of the subject to date and have provided a baseline for the task of reconciling more than 60 years of clinical documentation, with modern insights based on 21st century bacteriology and immunology.

I have collated the information in this booklet from the existing literature and my own recent clinical observation. Certain views are presented, particularly concerning aetiology, which would require extensive research to determine with any degree of certainty. Nevertheless, I hope that the current lack of research evidence will not prevent clinicians from using these materials on the indications tentatively outlined here. After assessing outcomes, I also hope that you will feed back your results in the form of published case studies or audit.

In the last section I have raised some research questions, which I feel could be taken up by clinical researchers. Over time, patterns of consistency are likely to emerge from our published case material. I am confident that a more compelling research agenda will follow as a matter of course.

Systems Thinking - Integrating past and current theory

One of the failings of Western medicine over the last century has been a tendency to over-rationalise illness around mechanistic models of causation. The clinical phenomena than emerge in states of illness can also be described in terms of complex dynamic reaction and counter-reaction.

The corollary to this 'systems-thinking', is a dawning awareness that complex illness cannot be cured merely by manipulating biochemical pathways with drugs. Treatments that deny the systems-context of illness will gradually become outdated as our biological sciences develop multi-variable models and a dynamic approach to the investigation of living phenomena. This shift in approach is already in evidence in modern ecological research.

Ecology and Micro-ecology

The relationships that exist:

- between individual organisms
- within and between groups of organisms and
- within and between species

are fundamental to biological co-existence and the sustainability of life on our planet. The issues of interdependence and competitive interplay operate at both micro-biological and macro-biological levels.

Micro-ecology of the Bowel

Much of this seminar will be concerned with a particular micro-macro biological relationship and its consequences for health: the symbiosis between the human organism and our bacterial flora.

Our animal evolution has involved unbroken contact with a shifting microbial environment. Aeons ago when the composition of the atmosphere was high in carbon dioxide and very low in oxygen, the open environment was populated with anaerobic microorganisms. Today the only natural habitat for anaerobes is within the intestine of animals. This vital symbiosis is a physiological and immunological necessity, which has required host adaptation and the evolution of specialised structures and organs.

When considered in embryological terms, the GI tract can be thought of as an outer body surface. As such, it represents a huge area of contact with the external environment - estimated at about 300 square metres when the area of the mucosal rugae and microvilli are taken into account.

In systems terms, immunity is no longer merely a defence from infection. Modern micro-ecology examines the relationship between microbe and host. This has been an issue of debate for more than one hundred years. It is a debate with which homeopathy has engaged in the past and needs to re-engage again. It is hoped that this seminar will help to bridge the historical gap, and allow medical homeopathy to re-enter the debate in an informed way.

Historical background

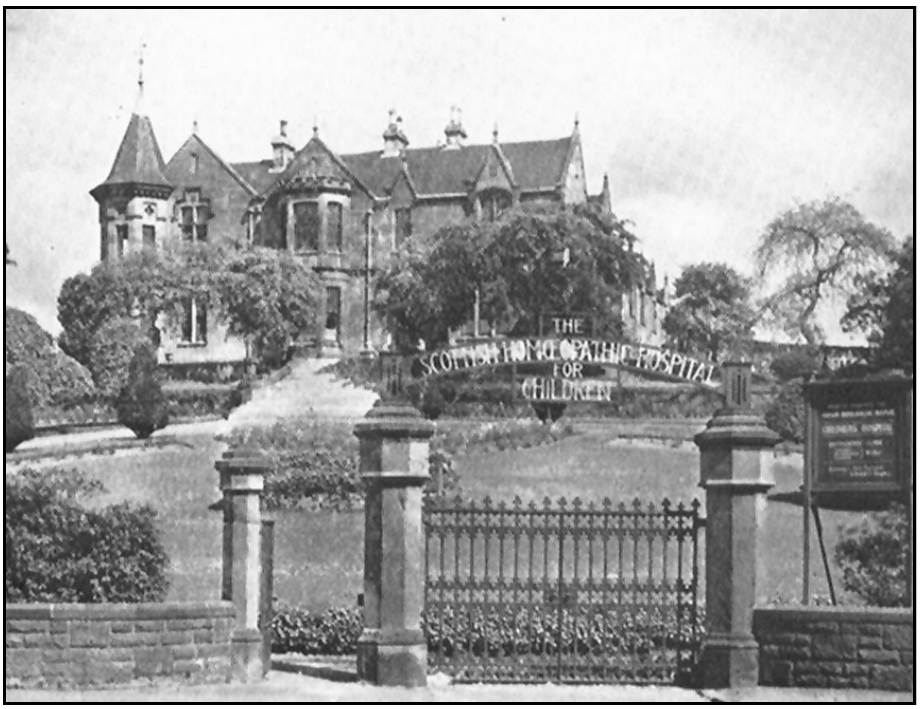
RLHH in 1930s.

Most of the seminal work on the bowel nosodes took place between 1920, when Edward Bach first introduced vaccine therapy to the homeopathic community[3] and 1960, when the British Homoeopathic Journal published a paper[18] containing details of over 30 years of John and Elizabeth Paterson's clinical work with the bowel nosodes.

The basic scientific investigation was exhaustive, although the technical methodologies used by Edward Bach and John Paterson have since been transformed by huge advances in bacteriological methodology [60 62].

Clinical testing was only possible because, in the middle years of the last century, a huge number of patients attended for homeopathy as their principle treatment choice. In the pre-NHS era, the Royal London and Glasgow Homoeopathic Hospitals had wide public support and were funded by public subscription. Also of vital importance was the involvement of several eminent consultants who collaborated with Bach and Paterson in the clinical testing of the medicines [12 13 17 18 26 29 30 31 32 35 37 39 40 41 43 44 46 50 52].

Charles Wheeler and Thomas Dishington require special mention for their input on the clinical indications and relationships [5 7 8 9 13] .
Many of the childhood indications [17 25] were worked out by clinicians at the Children's Homoeopathic Hospital in Glasgow under the guidance of John Paterson.



Former Scottish Homoeopathic Hospital for Children

We will begin with a general historical overview of bacteriology, before focussing more specifically on the development of the bowel nosodes.

Historical Overview

96 - 55 BC **Lucretius** recognized the existence of invisible "seeds" that were responsible for the transmission of disease. First recognition of contagion.

1677 **van Leeuwenhoek** - His major contribution to science was the production of simple single lens microscope through which he has able to see the major classes of bacteria, protozoa, etc. He described these observations in a series of letters to the Royal Society of London in which he described the little swimming creatures as "*animalcules*."

1750 **Spallanzoni** - First experiment to refute spontaneous generation which was the prevailing belief at the time.

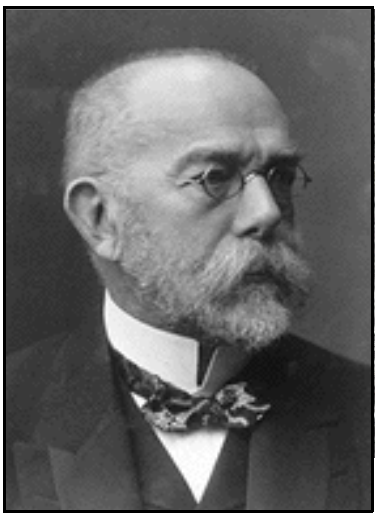
1800's **John Hunter** - provided direct evidence of disease transmission. He inoculated himself with purulent material from a man with gonorrhoea. Not only did he contract gonorrhoea, but as a bonus for his effort he also got syphilis!

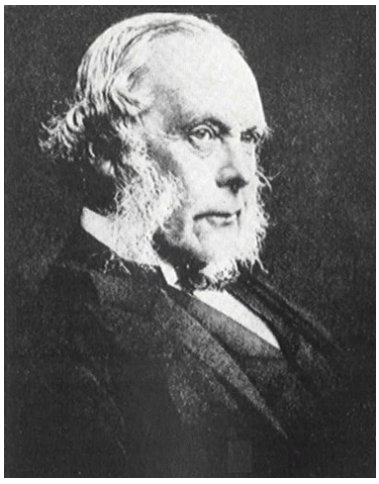
1796 **Edward Jenner** - introduced the concept of vaccination against small pox by inoculating individuals with material from lesions of a similar disease of cattle (cow pox).

1860 **Joseph Lister** - demonstrated the effects of antiseptics in surgery. Reduced the incidence of infections.

1864 **Louis Pasteur** - Used the now famous "swan neck" flask and once and for all laid to rest the concept of spontaneous generation. In the late 1800's Pasteur discovered methods of attenuation that were necessary for vaccine development

1867 **Robert Koch** - Provided the final evidence proving the germ theory. He established the etiologic role of bacteria in anthrax and as a result proposed a set of rules to be followed in the establishment of etiology. Because the etiologic agent of anthrax is a large rod-like organism (*Bacillus anthracis*) it was easy to observe by microscopy making it easier to identify.
The key to Koch's observation was the isolation of the organism in pure culture. While limiting dilutions could have been used (as described by Lister), Koch promoted the use of solid media (giving rise to separate colonies) and the use of stains.


Robert Koch


Joseph Lister


Louis Pasteur

1880 **Ebert** discovered *B. typhosus*

1882 **Koch** identified the tubercle bacillus and in so doing formalized the criteria of Henle (1840) for distinguishing pathogenic microbes. This set of criteria is known as Koch's postulates. The postulates state that:

1. The organism is regularly found in the lesions of the disease
2. It can be isolated in pure culture
3. Produces a similar disease in an animal (model)
4. Organism can be recovered from lesions in the model

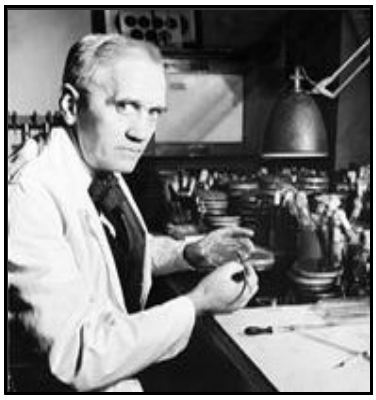
1883 **Koch** discovered the *cholera vibrio*

1883 **Klebs** and **Loeffler** discovered the *diphtheria* bacillus

1886 **Frankel** discovered *pneumococcus*

1887 **Weichslebaum** discovered *meningococcus*

1889 **Kitasato** discovered *tetanus bacillus*. In 1889, Kitasato also discovered tetanus toxin. These observations led others to believe that disease should be caused by cell-free solutions containing toxins. This almost became the fifth of Koch's postulates.

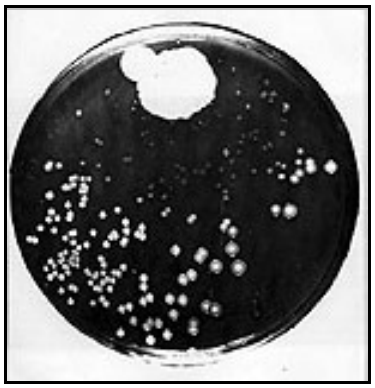


Alexander Fleming

1894 **Yersin** discovered the cause of the plaque Initial identification of toxic substances from microbes began in 1888 when Roux and Yersin showed that cell-free solutions of *diphtheria* bacillus caused symptoms similar to the disease.

1904 **Paul Ehrlich** - pioneered the area of chemotherapy. Used dyes active against *trypanosomes* and arsenicals active against *spirochetes*.

1919 **Edward Bach** - bacteriologist - joins the staff of the Royal London Homeopathic Hospital and begins to investigate the effects of autologous vaccines.



Fleming's famous penecillin plate

1925 **Fleming** reported on the identification of a *Penicillin notatum* colony that lysed *Staphylococci*.

1927 **John Paterson** establishes a bacteriology laboratory in Glasgow and begins to investigate the bacteriological effects of homoeopathic treatment.

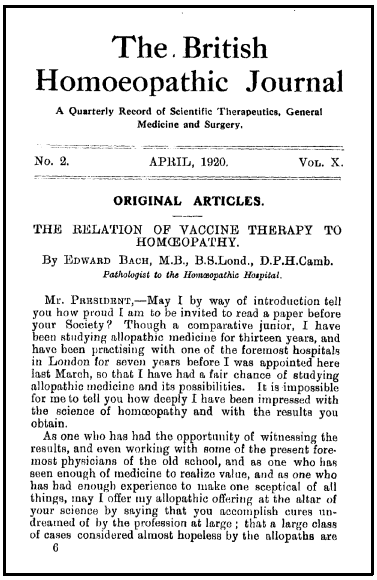
1935 **Domagk** discovered the first active sulfa

1939 **Chain** purified penicillin

1944 **Waksman** discovered streptomycin

1960 **Elizabeth Paterson** collates together the findings of John and Elizabeth Paterson and publishes in the BHJ.

Dr. Edward Bach


The British Homoeopathic Journal

A Quarterly Record of Scientific Therapeutics, General Medicine and Surgery.

No. 2. APRIL, 1920. Vol. X.

ORIGINAL ARTICLES.

THE RELATION OF VACCINE THERAPY TO HOMŒOPATHY.

By Edward Bach, M.B., B.S.Lond., D.P.H.Camb.
Pathologist to the Homœopathic Hospital.

Mr. President,—May I by way of introduction tell you how proud I am to be invited to read a paper before your Society? Though a comparative junior, I have been studying allopathic medicine for thirteen years, and have been practising with one of the foremost hospitals in London for seven years before I was appointed here last March, so that I have had a fair chance of studying allopathic medicine and its possibilities. It is impossible for me to tell you how deeply I have been impressed with the science of homœopathy and with the results you obtain.

As one who has had the opportunity of witnessing the results, and even working with some of the present foremost physicians of the old school, and as one who has seen enough of medicine to realize value, and as one who has had enough experience to make one sceptical of all things, may I offer my allopathic offering at the altar of your science by saying that you accomplish cures undreamed of by the profession at large; that a large class of cases considered almost hopeless by the allopaths are

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Edward Bach was born in 1886 in Birmingham and qualified at University College Hospital, London, in 1912. After house appointments he became assistant bacteriologist at UCH. Edward Bach joined the staff of the Royal London Homeopathic Hospital in 1919, and quickly recognised some parallels between vaccine therapy and homeotherapeutics. His initial thoughts were published in The British Homoeopathic Journal, in April 1920. His introduction is given below. Bach's earliest observations still have resonances in modern homeopathic practice and are outlined on the pages that follow.

THE RELATION OF VACCINE THERAPY TO HOMOEOPATHY.

By Edward BACH, M.B., B.S.Lond., D.P.H.Camb.

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Mr. PRESIDENT,

May I by way of introduction tell you how proud I am to be invited to read a paper before your Society. Though a comparative junior, I have been studying allopathic medicine for thirteen years, and have been practising with one of the foremost hospitals in London for several years before I was appointed here last March, so that I have had a fair chance of studying allopathic medicine and its possibilities. It is impossible for me to tell you how deeply I have been impressed with the science of homoeopathy and with the results you obtain.

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The five key points in this paper are outlined in the pages that follow:

The 'Orthodox' Concept	The 'Homeopathic' Concept
1. Microdose phenomena (vaccines)	Principle of the *Minimum Dose*
2. Cross antigenicity	*Principle of Similars*
3.'Systems effects' in illness involving the mind	The *Symptom Picture*
4. Immunology - Infective triggers to illness	The *Aetiological nosode* and '*Miasms'*
5. Use of autologous vaccines in chronic disease	The *Bowel Nosodes*

Edward Bach - Key points from

The Relation of Vaccine Therapy to Homeopathy, 1920

Edward Bach

1. Microdoses and the ***Principle of the Minimum Dose***

Vaccines contain an estimated $^{1}/_{200000}$ mg of bacterial substance at a concentration comparable with a 7x or 8x potency of arsenic.- Bach [3]

In the pre-antibiotic era, vaccine therapy was used in the treatment of acute infection. The practice was to stimulate host resistance during infection with *microdoses* of the infecting organism. This has parallels with *isopathy*. Unfortunately, modern hospital medicine is 'evidence constrained' in it's approach to serious multi-resistant infections and fails to re-employ these techniques when anti-microbials fail.

In typhoid 500 or 1000 million bacilli are given as prophylaxis. But in treating a patient with the disease, a hundredth or even a thousandth of that would be used. - Bach [3]

Dose-response remains an issue for us today in pharmacology, homeopathy and immunology

2. Cross antigenicity and the ***Principle of Similars***

Organisms closely allied to the causative germ of a particular disease may give benefit when used as a vaccine...

Thus any of the large numbers of varieties of streptococcus will be beneficial in an illness with a particular streptococcus...

Immunising with typhoid organisms produces a certain amount of resistance to paratyphoid and other closely allied bacilli. The blood of patients who have had typhoid or who have been inoculated with it will agglutinate the sera of dysentery or paratyphoid bacilli. - Bach [3]

The concept of cross antigenicity had been 'known' ever since Jenner recognised that the skin of milk-maids showed no signs of small-pox scarring.

Case: (G005) Boy aged 9: asthma since Mycoplasma pneumonia.

Patient: Active: many hobbies - scouts, chess, cycling, tennis ...

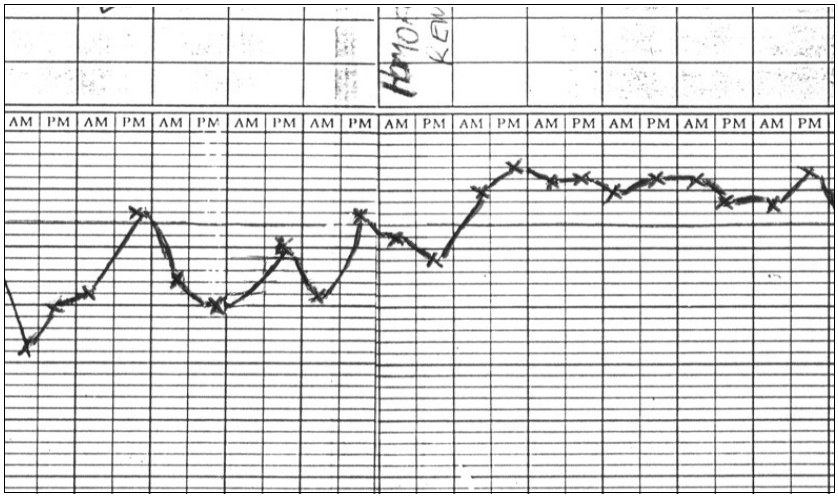
Desires milk

Desires bananas

Grandfather died of TB.

Treatment:

Tub bov. 30c (*Mycobacterium tuberculosum bovinum*) - highly successful.



Mycobacteria - their potential rôle in immunotherapy for atopy

'... a possible explanation for the protective effects of exposure to bacteria or their products in early life, when sensitisation occurs, is their action to increase production of interferon (. This concept has given rise to the 'hygeine hypothesis' in which changes to infant diets, early use of antibiotics, and reduced exposure to bacterial products predispose to the persistence of Th2 responses in childhood. It follows that one approach to treating allergy would be to take advantage of the capacity of mycobacteria to evoke strong production of interferon (, possibly with the soil saprophyte *Mycobacterium vaccae* since this is not a human pathogen. Clinical trials of this 'vaccine' for rhinitis and asthma are in progress, and the early results are promising.'Holgate,S.T. *Allergic disorders* Science, medicine, and the future. BMJ Vol.320 22 Jan.2000 pp 231-4

Shirikawa T, Enomoto T, Shimazu SI, Hopkin JM *The Inverse association between tuberculin response and atopic disorder.* Science 1997; 275: 77-9.

Martinez FD, Holt PG. *Role of microbial burden in aetiology of allergy and ashthma*. Lancet 1999; 354 (paediatrics suppl. II): SII 12-5.

Wang C-C, Rook AW. *Inhibition of an established allergic response to ovalbumin in BALB/c mice killed by Mycobacterium vaccae.* Immunology 1998;93:307-13

3. Awareness of 'systems effects' in illness - ***Symptom Picture***

Individuals having unusual fears, such as dread of fire, heights, crowds, traffic, have almost invariably an organism of the paratyphoid group of bacillus. The highly strung, nervy person with anxious expression, often with a fixed look, frequently has a bacillus of the Proteus group. the patient who at a casual glance seems to be in perfect health and yet has some serious chronic disease such as tubercle, often has organisms of the Coli mutabile group. The folk who bruise and bleed easily generally posses a dysentery type germ. - Bach [3]

This is the first documented description of the mental picture in post infective and/or 'carrier' states. It is difficult to assess whether these are Bach's true observations, or whether they are included in recognition of the Kentian preoccupations of his audience. These early thoughts on mind symptoms are reflected in later papers by other authors, where they have been incorporated into the *materia medica* data of the nosodes themselves.

Clinical Cases:

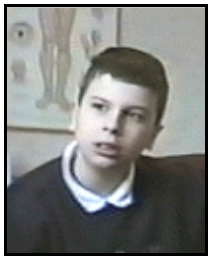
Girl aged 8.

Recurrent UTI (most frequently with E.Coli.) Many episodes usually treated with antibiotics. The onset of each infective episode of is associated with marked changes in mood and behaviour before the onset of urinary symptoms. Treated successfully with *Chininum sulph.* and *Mutabile*.

Boy aged 4.

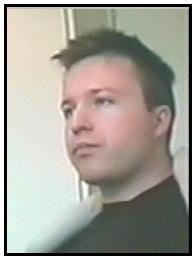
Developmental delay and retardation due to congenital anomaly. Hypospadias: two failed surgical repairs due to wound breakdown. Recurrent UTI, heralded by irritability and disturbed sleep patterns. Marked reduction of incidence in infections following *Baryta carbonica*. Following surgery is successful with uncomplicated wound healing.

4. Infective triggers in disease and immunomodulatory phenomena associated with immunisation - ***Pathography*** and the ***Aetiological Remedy***



In cases of rheumatoid arthritis and neuritis, I have several times [after vaccination] *seen pains come out during the* [primary response], *which the patients state they have not had since childhood.* - Bach [3]

Case 11 (D084) has a re-evocation of past symptoms with every vaccination or episode of tonsillitis.



It is surprising in cases of chronic disease how, after two or three doses of a vaccine obtained from a single organism in the intestine, the whole condition improves and the patient becomes well. - Bach [3]

Case 16 (T056): complete resolution from 3 doses.

Infective Triggers in Disease - continued

Having saved your patient [with pneumonia] with an inoculation of pneumococci or streptococci made from the sputum, after convalescence it is well to find the intestinal organism and give doses which will raise the general resistance against disease in all forms. - Bach [3]

In orthodox western medicine we no longer treat infection with vaccination, but we continue to recognise problems with full recuperation in a significant number of cases. Orthodox medicine has very few solutions for those failing to recover, after the pathogen has been 'eradicated'.

Post Infective States

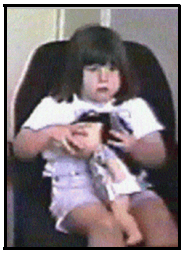
Those case studies provided, which demonstrate a clear infective aetiology in their chronic state, include numbers: 1, 2, 8, 11,16,19, 21, 25, 35. Cases with signs of a likely infective component in their aetiology include numbers 5,6,7,10,17... (See Illustrative Cases)

'Isopathic' treatment based on Post-Infective Aetiology

In his recognition of post-infective sequelae Bach predates the later concept of post infective dysbiosis. He also recognises that post-infective states are often amenable to treatment with a nosode of the causative agent.

Case (C130) Girl aged five - asthma (of 18 months duration) resolves permanently within a few days of *Pertussin* 30c. (Yorkhill children's hospital recognised the trigger aetiology but had been treating with inhaled steroids and bronchodilators)

Case (JM) Girl aged seven - chronic cough, ENT catarrh and tendency to mouth breathing improves after *Morbillinum* 30. (Reacted aversely to measles vaccination in the past.)

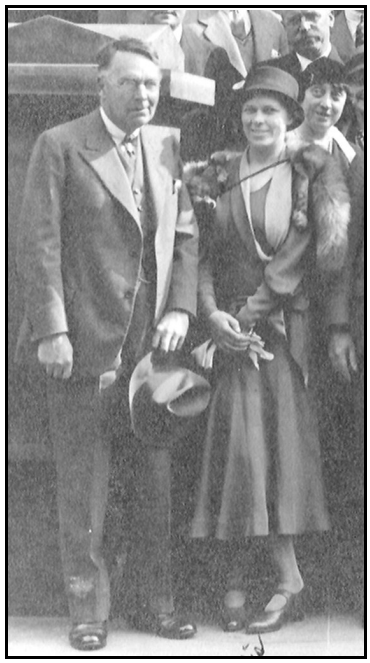


5. The Use of Autologous Vaccines in Chronic Disease

The potential role of autologous vaccines [3 4 9] in chronic disease was investigated by Bach (bacteriologist) and Wheeler (clinician) who, under joint authorship, published a treatise in 1925 entitled. Chronic Diseases - a working hypothesis [7]. In this paper they selected 500 cases, which had been under observation for at least six months and had received no treatment other than the autologous vaccine. The results are given in Table 2 below.

No. of cases	Diagnosis	Result of treatment			
		Excellent	Good	Moderate	Fail
11	Chronic skin	-	6	4	1
27	Anaemia	2	19	6	-
5	Bacilluria	2	2	1	-
43	Chronic rheumatism	7	32	3	1
3	Lumbago	1	2	-	-
1	Fibrositis	-	-	-	1
77	Rheumatoid arthritis	9	29	31	8
16	Sciatica	5	6	5	-
6	Neuritis	2	2	2	-
12	Epilepsy	-	8	3	1
33	Chronic headache	6	15	10	2
87	Neurasthenia	11	54	19	3
5	Hysteria	-	2	3	-
3	Insomnia	1	2	-	-
7	Mania	1	4	1	1
7	Graves Disease	-	7	-	-
2	Hyperpiesis	1	1	-	-
1	Alcoholism	-	1	-	-
17	Chronic gastritis	5	10	1	1
38	Chronic colitis	6	18	13	1
3	Constipation	-	1	2	-
5	Cholecystitis	-	4	1	-
12	Chronic catarrh	2	7	3	-
9	Asthma	-	7	2	-
3	Chronic bronchitis	-	3	-	-
1	Emphysema	-	-	-	1
32	Malignancy	20 (for pain)	9	3	-
6	Gout	-	3	3	-

Table 2



The picture above shows Charles Wheeler and Marjory Blackie at a Homeopathic Congress in Glasgow, (1933). *Chronic Diseases - a working hypothesis*[7], was the conceptual stimulus for the development of the bowel nosodes over the following 30 years.

Bach's vaccines were prepared from non-lactose fermenting bacilli. The culture was incubated for 16h. It was then washed in 6cc of normal saline and killed by immersing the sealed tube in water at 60 degrees for 1h. the emulsion was then diluted to 25ml with saline, and 0.25ml of a 1 in 4 mixture of Lysol in alcohol was added. There were three classes of bacteria depending on whether they produced acid and/or gas in glucose and/or saccharose. These were prepared from about two hundred cases for each class of bacterium, mixed in a 400ml.

Dosage of both polyvalent and autogenous vaccines commenced with 0.04 ml rising to 0.9 ml for the sixth injection. the final injection could be as much as 2ml. Where there was a severe reaction the following dose was not increased in volume.

Thomas Dishington and **John Paterson**

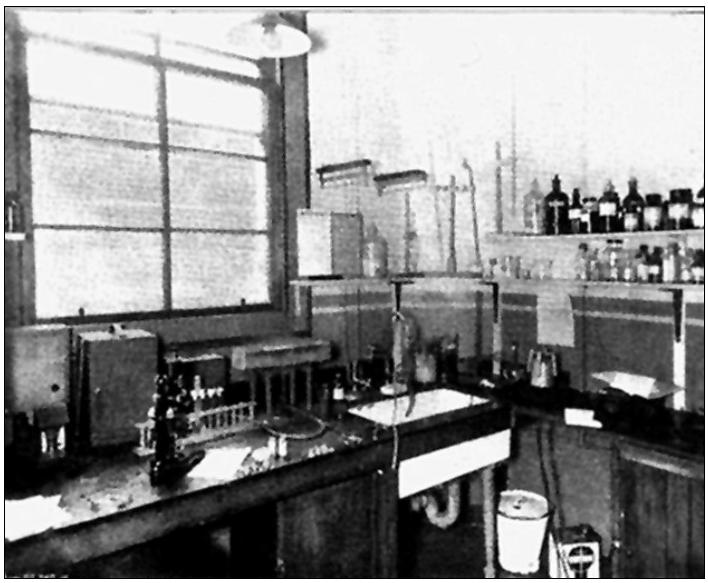
"The late Thomas Dishington conceived the idea of having the autologous vaccines of patients potentised and from a collection of autologous vaccines making a proving." - Bach

Quoted from John Paterson - *Indications for the use of Intestinal Nosodes in Diseases of Children.* [25]

The Bach Polyvalent Nosode was a potency prepared from the intestinal vaccine already in existence [14] .

In association with Thomas Dishington, John Paterson made clinical observations on the effects of the polyvalent nosode at the Scottish Homeopathic Children's Hospital and by 1927 they were satisfied that the nosode had clinical value. Wheeler, Bach and Dishington published a conjoint clinical paper [8] in 1927.

After presenting their paper at the Quinquenial International Homeopathic Congress of 1927. Paterson visited Bach and learned his bacteriological techniques, prior to establishing a bacteriology laboratory in Glasgow.



Laboratory at the old Glasgow Homoeopathic Hospital

OUTFIT FOR COLLECTING SPECIMENS (Paterson) [21]

(1) Hard glass test tube 6 inches x 3/8 inches.

(2) Cork (must stand 160 degrees dry heat.)

(3) Nickel probe with:

(a) Sharpened end - for insertion into cork.

(b) Roughened end - carries cotton wool tip.

(4) Label for patient's name, address, etc.,

(5) Wooden container - for transit.

Bowel swabs are sterilised by dry heat 160 °C. for 30 minutes.

TAKING SPECIMENS. [21]

(l) Use clean dry receptacle to receive faeces.

(2) Withdraw cork from glass tube.

(3) Do not touch metal probe or cotton, wool tip with fingers.

(4) Dip point into the faecal material so as to soil cotton

(5) Shake off any loose pieces and replace probe into tube.

(6) Replace tube, firmly corked, into wooden container ready for transit.

Bulk specimens of faeces are not required and are unsuitable unless for immediate culture. Specimens taken by "bowel swab" retain their primary characteristics for long periods without change. Secondary change may occur in bulk faeces retained for any length of time.

Bacteriological Method

John Paterson

The laboratory method used by Bach and Paterson was based on the standard bacteriological method of their day.

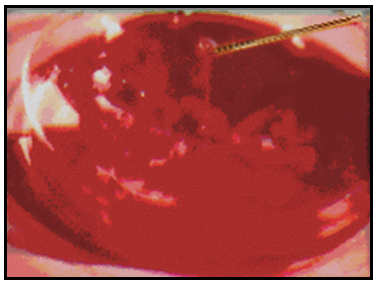
After collection and transport the specimen was plated and analysed as follows (from Paterson [23]):

Emulsify add 5 ml distilled water to original tube, and use contaminated cotton wool and probe.

Inoculate MacConkey* Plate by spreading with sterile bent glass rod - a small drop of emulsion from tip of probe.

Incubate at 37.5 °C. for 18 hours.

Examine by transmitted light (daylight lamp) for distinctive non-lactose colonies



Sub-culture to plain agar slope a well-isolated non-lactose colony.

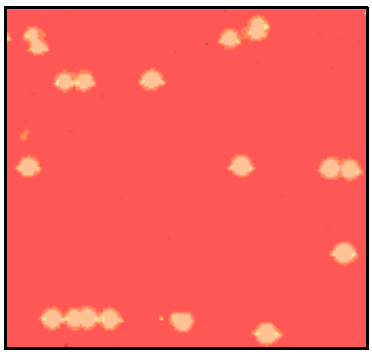
Incubate for 18 hours.

Identify by inoculation into selected sugar solutions.

If sub-culture if required for autogenous vaccine it should be prepared immediately by:

1. Washing surface and emulsifying with 5 c.cs of distilled water.
2. Sealing in test tube and heating in water-bath at 60C. for 30 minutes.

*This medium inhibits growth of gram positive bacteria, and allows easy differentiation between organisms which ferment lactose and tose that do not. On MacConkey agar, colonies of lactose fermenting organisms are brick red in colour, while those of the non lactose fermentors are uncoloured and transparent or whitish.



Non-lactose fermenting colonies

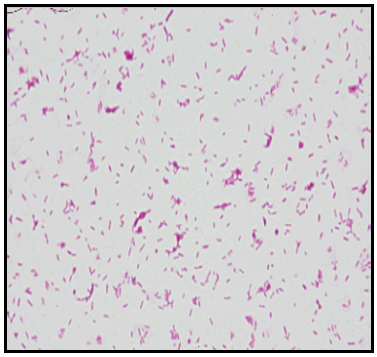
Bacteriology of the Bowel - Normal flora

99.9% of the normal flora consists of anaerobes numbering 10^{11}/g of faeces. The major bacterial groups in intestinal microflora are roughly divided into the following three groups:

1) the strict anaerobic group, including *Eubacterium*, *Bacteroides*, *Fusobacterium* and *Clostridium*,

2) the anaeobic lactic acid-producing bacteria, including *Bifidobacterium*, *Lactobacillus* and *Enterococcus*,

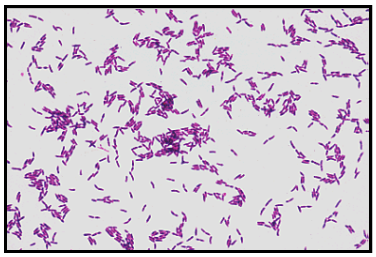
3) facultative anaerobic bacteria, incl. *enterobacteriaceae*. 10^{8}/g of faeces.



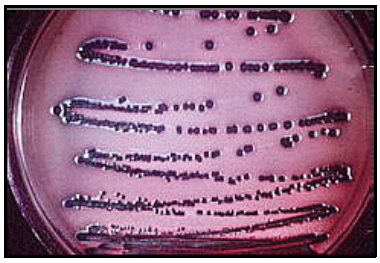
Bacteroides

Table 3 . Normal Faecal Flora

ALWAYS PRESENT	
Genus	Example
Bacteroides	*Bacteroides fragilis*
Clostridium	*Clostridium perfringens*
Enterococcus	*Enterococcus faecalis*
Escherichia	*Escherichia coli*
FREQUENTLY PRESENT	
Genus	Example
Aeromonas	*Aeromonas hydrophila*
Acinetobacter	*Acinetobacter baumani*
Alcaligenes	*Alcaligenes faecalis*
Bacillus	*Bacillus subtilis*
Candida	*Candida albicans*
Corynebacterium	*C. pseudodiph.*
Enterobacter	*Enterobacter aerogenes*
Hafnia	*Hafnia alvei*
Klebsiella	*Klebsiella pneumoniae*
Neisseria	*Neisseria sicca*
Peptostreptococcus	*P.asacharolyticus*
Pseudomonas	*P. aeruginosa*
Proteus	*Proteus vulgaris*
Providencia	*Providencia rettgeri*
Sachromyces	*Sacharomyces cerevisiae*
Staphylococcus	*S. epidermidis*
Streptococcus	*viridans group*



Eubacterium



E.Coli plate

Bacteriology of the Bowel - Enterobacteriaceae

Proteus mirabilis

This is a large family of organisms containing several genera - usually associated with the intestinal tract. Many members are considered to be normal flora. The characterisitics of the enterobacteriaceae are as follows:

A. Gram-negative, Rods

B. Facultative

C. Can be motile with peritrichous flagella

D. Ferment glucose

E. Oxidase negative

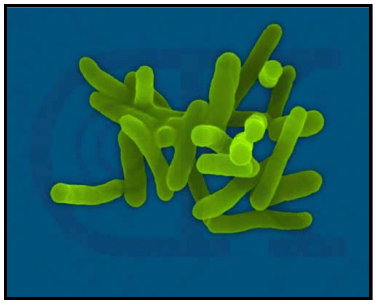
F. Can reduce NO_3 to NO_2

G. Produce O, K, and H antigens

O = the outer part of LPS

K = capsule

H = flagella



Shigella

Among the *enterobacteriaceae* are a number of enteric pathogens:

- *Escherichia coli* [2]
- *Salmonella* spp.
- *Yersinia enterocoltica*
- *Shigella* spp.

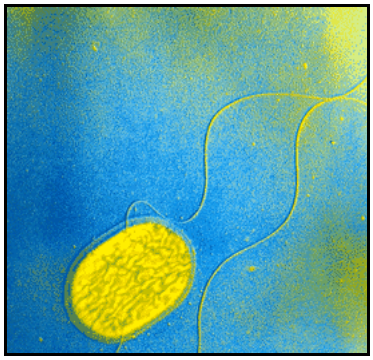
Some of the *enterobacteriaceae* are recognised as extra-enteric pathogens:

- *Escherichia coli* [2]
- *Klebsiella pneumoniae* [1]
- *Klebsiella oxytoca* [1]
- *Proteus mirabilis* [1]
- *Enterobacter aerogenes* [1]
- *Enterobacter cloacae* [1]
- *Serratia marscens*
- *Salmonella*
- *Citrobacter*
- *Edwardsiella*
- *Morganella*
- *Provedencia* [1]

[1] Frequently present in the stool of healthy subject

[2] *E. coli* subtype(s) always present - wide range of pathogenicity

Bacteriology of the Bowel -



Salmonella

Potentially Pathogenic Microorganisms (PPMOs)

The influence of antimicrobial agents [and systems stressors?] *on the concentrations of PPMOs (potentially pathogenic micro-organisms) in the bowel is determined both by their influence on the flora that provides colonisation resistance (CR) and by their direct influence on PPMOs.*

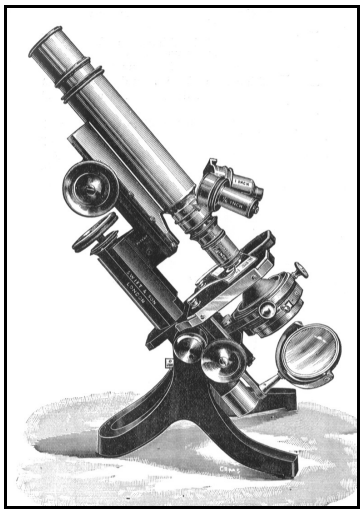
M. Delmée *Antibiotics*

The pathogenic status of the most important enterobacteria are given below. Their corresponding bowel nosode(s) are listed. The apparent uncertainty about these is explained overleaf.

enterobacteriaceae	**pathogenic status**	**bowel nosode(s)** **(by fermentation profile)**
Shigella spp.	EP	Dysenteria co.
Escherichia coli	EP / PPMO	??? Mutabile
Salmonella spp.	EP	Gaertner ?? Mutabile
Klebsiella spp.	PPMO	?
Proteus spp.	PPMO	Proteus
Enterobacter cloacae	PPMO	?? Bacillus 7
Citrobacter	PPMO	? Bacillus 7
Edwardsiella spp.	PPMO	? Morgan-p. ??? Proteus
Morganella	PPMO	Morgan-p. ? Mutabile

Table 4

Identification of the bowel nosodes.



Clearly, most of the information on normal bowel ecology, outlined on the previous page, was unknown when Bach and Paterson began their investigations into non-lactose fermenting bacteria. They used direct microscopy and four standard sugar-fermentation tests to distinguish between the isolated cultures.

In modern terms this methodology is inadequate on its own to properly identify the different bacterial populations. The implications of this will be discussed in the next chapter.

Table 5 below shows the fermentation characteristics of the non-lactose groups that they isolated.[21] The production of acid/gas, on fermentation of glucose, lactose, saccharose and dulcitol is recorded below.

	18 hours		24-30 hours			
Lactose	a/g	-	-	-	A/G	A/G
Glucose	-	A/G	A/G	A/G	A/G	A/G
Dulcitol	-	-	-	A/G	-	A/G
Saccharose	-	-	-	-	-	-
	No.10	*Morgan co.*	*Morg-p.*	*Morg-g.*	*Mutabile*	*No 10*

Table 5a.

	18 hours				24 - 30 hours	
Lactose	-	-	-	-	-	-
Glucose	A/G	A/G	A/G	A	-	-
Dulcitol	A/G	-	A/G	(A)	-	-
Saccharose	A/G	A/G	-	-	-	-
	No. 7	*Proteus*	*Gaertner*	*Dys.*	*Faecalis*	*Sycotic co.*

Table 5b.

The nosode was prepared by covering the pure-culture isolates with a film of sterile water for 18 hours, collecting the solution and heat-treating it in sealed tubes, and then potentising.

Limitations of Bach and Paterson's methodology

Sugar tests with peptone water are still used to a limited extent in modern bacteriology, but the original batch of four are now considered insufficient to distinguish between pathogenic and non-pathogenic strains of bacteria (including the common bowel organism *E. coli* which is known to have many thousands of biotypes.)

Modern analysts also recognise that species and biotypes merge into one another rather than forming distinct entities. Some strains of *E.coli* and *Shigella*, for example, are closely related antigenically and biochemically.

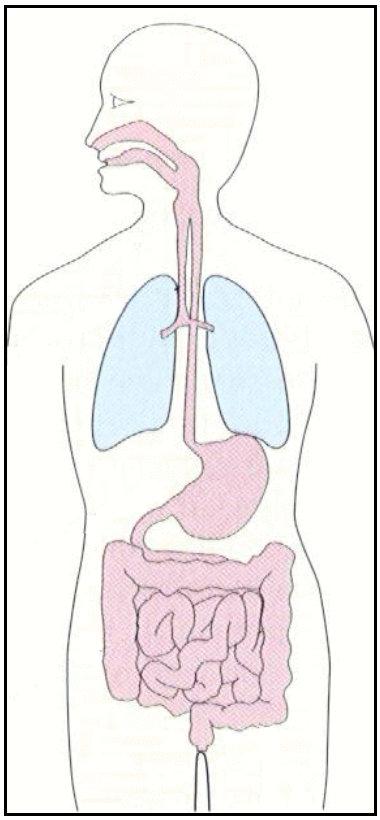
Definitive typing of bacteria today may involve a computerised analysis of several hundred biochemical test results and genetic 'fingerprinting.'

If we restrict ourselves to the results of Bach and Paterson's fermentation tests [21], we can see that there is considerable uncertainty concerning the bacteriology of the nosodes. The microscopy and naked eye appearance of the colonies, go a little way to reducing this uncertainty.

Nosode	Bacteria conforming to Bach and Paterson's test types
B. Morgan	Morganella morganii, Proteus mirabilis (4% sucrose +ve), Salmonella subgenus IV, Aeromonas salmonicida, Edwardsiella tarda, Escherischia blattae, Haffnia alvei (7% are lactose +ve)
B. Gaertner	Salmonella paratyphi A, Salmonella subgenus I & II, Salmonella cholerasuis (27% are dulcitol positive)
B. No. 7	Citrobacter koseri (45% dulcitol +ve, 85% lactose +ve, 45% sucrose +ve) Enterobacter cloacae (12% dulcitol +ve, 76 lactose +ve)
B. Proteus	Edwardsiella hoshinae, E. tarda biogroup 1, Obesumbacterium proteus biogroup 2, Proteus myofaciens, Proteus penneri, Proteus vulgaris biogroup 2.
B. Mutabile	Morganella morganii (lactose +ve 3%), Salmonella subgenus III (lactose 33% +ve) Salmonella subgenus III (lactose +ve 33%), Salmonella subgenus IV (lactose -ve)
B. Dysenteriae	Shigella dysenteriae, Shigella flexneri, Shigella boydii, Salmonella gallinarium, Salmonella typhisus
B. Faecalis	An enterococcus ? , Bacillus faecalis alcaligenes, ? Acinetobacter
B. No. 10	?

Table 6 - reproduced from Alexander, M. *Reidentifying the bowel nosodes* BHJ April 1998 Vol 77. pp. 67-71

Micro-ecology of the Human GI Tract - Introduction



An estimated10^{14} living bacteria encompassing more than 400 species are present in the human gut. This represents a very complex milieu which is metabolically active. Wide variation occurs because the intestinal ecosystem has a close relationship with the host and which is perpetually in contact with xenobiotics, mainly food and drugs.

The microflora of different regions of the GI-tract:

The stomach normally contains a small number of predominantly Gram-positive and aerobic bacteria and the small intestine represents a transitional zone between the scarce population of the stomach and the high density population of the colon.

In the proximal small bowel bacterial concentration and pattern of microflora are similar to that of the stomach, while in distal ileum gram-negative begin to outnumber the gram-positive and bacterial concentration increases significantly.

The small intestine contains only 10^{3} to 10^{5} bacteria per gram of luminal content. The forceful peristalsis exceeds the bacterial rate of multiplication and only bacteria which adhere to the mucosa can persist.

The most dramatic change occurs across of ileo-cecal valve, with the total number of micro-organisms increasing up to one million fold, and anaerobes outnumbering aerobes in the ratio of 1000:1.

In the colon, peristalsis is slower and bacterial population reaches immense numbers: 10^{9} to 10^{11} per gram. Concentration of nutrients are poor in the lumen but richer near the epithelial cells where mucus and micronutrients are concentrated. Bacterial factors promoting colonisation of the mucus layer result in enhanced metabolism and multiplication.

Development of the human intestinal microflora:

The human foetus is devoid of bacteria before birth. After birth it rapidly becomes colonised. By the end of the second year, the composition of the child's microflora resembles closely that of the adult. The composition of the adult flora changes with age.

Disturbances in the intestinal microflora:

Although the composition of the GI-tract flora is fairly stable in healthy persons, it can be altered by many factors such as antibiotics, emotional stress, surgical operations of the GI-tract, disorders of peristalsis, inflammatory bowel diseases, cancer, disorders of the cellular and humoral immune system and age.

Micro-ecology of the Human GI Tract - Research models

The modern concept of the intestinal ecosystem is largely based on studies by Schoedler and Dubos (1964).Various additional models have evolved over the past 30 years, through the efforts of several research teams in different parts of the world (Luckey-Savage-Freter-Midtvedt-Raibaud-Ducluzau etc).

Studies, based on the use of germfree animals and gnotobiology as well as new culture techniques for anaerobic bacteria have provided research models and have led to a number of new concepts, as well as opening up several new avenues for future research.

Regulation within the Intestinal Ecosystem

Our bacterial population is maintained by a surface immunity based on processes of:

- recognition
- biofeedback (immunological and biochemical)
- local environmental shift involving synergy / inhibition
- modulation of the competitive balance between sub-populations

Recognition in the gastrointestinal tract involves the gut-associated lymphatic tissue (GALT) which 'learns' by pinocytosis and perabsorbtion of bacteria and bacterial products.
The stability (and protective capacity) of the immune system as a whole is dependant on the maintenance of dynamic equilibrium within the healthy gut.

Disturbances of Symbiotic Equilibrium

If gut symbiosis is rendered chaotic, under the influence of toxins, pathogens, drugs, or psycho-neuro-endocrine stress, then immunological chaos will manifest not only in the GI tract but elsewhere giving rise to a globally lowered resistance to opportunistic infection.

Bacterial sub-populations change in their relative numbers and locations with changes in the environment. In the 1950s Baumgaertel suggested that deleterious changes in the environment ('milieu' or 'terrain') could result in coliforms degenerating into toxic variants.

Neustaedter found that enterobacteria subtypes (usually E. coli), which ferment lactose very slowly, were clearly associated with chronic illness.

Haenel introduced the terms of ***eubiosis*** and ***dysbiosis.*** The term eubiosis is used to describe balanced steady conditions between the microflora and the host, whereas dysbiosis means a significant shift away from eubiosis.

Various dysbiotic states are recognised. Most typically they manifest as repression of *bifido-bacteria* by tribes of *colifoms*, *enterobacteria*, and especially an increase of *enterococci*, *proteus*, yeasts and *clostridiae*.

Micro-ecology of the Human GI Tract - Eubiosis

In the eubiotic state faecal flora is:

- 90% obligate anaerobic *bifido-bacteria* and *bacteroides,*

- 10% obligate aerobic bacteria (*coliform, enterococci, lactobacilli*),

- less than 1% of other enterobacteria
(*proteus, clostridiae, staphylococci,* aerobic spores and yeasts).

There is evidence to indicate that patterns of illness (distinct from illness caused by obvious infection by enteropathogens) are associated with characteristic shifts in the microflora.
For example: increase in *Group D Streptococci* in faeces of ulcerative colitis patients. In other studies it has been shown that colonic flora changes in composition when the patient is under stress.

For many years it has been standard practice in stool microbiology to identify the relative populations of obligate and facultative anaerobes. Identification of enterobacteria (particularly *E. coli*) was also based on differentiation of lactose fermenting degree (1-3)

Group 1
(very good fermentors) are frequently found in healthy subjects.

Group 3
(inhibited or delayed fermentation) are frequently found in sick patients.

This has some consistency with the findings of Bach and Paterson although they focussed on the balance between good fermentors and non-lactose fermentors (NLF). Their singular emphasis on the importance of non-lactose fermenters (NLF) is not fully consistent with observations of Seeliger and Neustaedter, who did not particularly associate NLF with illness.

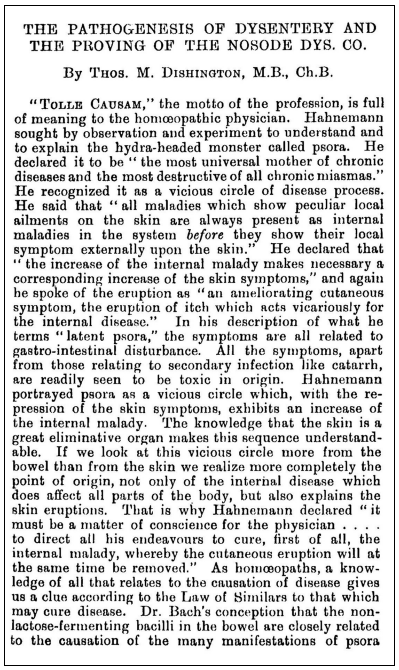
Bach and Paterson, however, were able to demonstrate changes in the bowel flora under the influence of homeopathic treatment. They found that, in chronic cases, the bowel flora changed temporarily after homoeopathic treatment, to a state which they normally associated with illness (increased NLF).

At first glance this seems to be a paradoxical response to treatment. One possible explanation for the phenomenon may concern changes in the surface adherancy of specific subpopulations, their displacement and their expulsion via the stool.

Electron microscopy has revealed the intimacy of the surface adhesion of many organisms on the microvilli. Some species have highly adapted surface protein structures which facilitate adhesion, in some cases with a degree of damage to the microvillous structure of the endothelium. An appropriate homeopathic stimulus may ‘loosen’ their grip on the endothelium and allow safer organisms to populate the vacated areas.

Homeopathic Concepts and The Influence of Remedies

In the following section we will consider Paterson's observations of the changes in the bowel flora and reconsider the interpretation of these observations. Before we examine current ideas on intestinal dysbiosis we should consider the 20th century perspective. One of the most lucid historical sources on the subject is Thomas Dishington's paper *The Pathogenesis of Dysentery and the Proving of the Nosode Dys. co.* [13]



THE PATHOGENESIS OF DYSENTERY AND THE PROVING OF THE NOSODE DYS. CO.

By Thos. M. Dishington, M.B., Ch.B.

"Tolle Causam," the motto of the profession, is full of meaning to the homœopathic physician. Hahnemann sought by observation and experiment to understand and to explain the hydra-headed monster called psora. He declared it to be "the most universal mother of chronic diseases and the most destructive of all chronic miasmas." He recognized it as a vicious circle of disease process. He said that "all maladies which show peculiar local ailments on the skin are always present as internal maladies in the system *before* they show their local symptom externally upon the skin." He declared that "the increase of the internal malady makes necessary a corresponding increase of the skin symptoms," and again he spoke of the eruption as "an ameliorating cutaneous symptom, the eruption of itch which acts vicariously for the internal disease." In his description of what he terms "latent psora," the symptoms are all related to gastro-intestinal disturbance. All the symptoms, apart from those relating to secondary infection like catarrh, are readily seen to be toxic in origin. Hahnemann portrayed psora as a vicious circle which, with the repression of the skin symptoms, exhibits an increase of the internal malady. The knowledge that the skin is a great eliminative organ makes this sequence understandable. If we look at this vicious circle more from the bowel than from the skin we realize more completely the point of origin, not only of the internal disease which does affect all parts of the body, but also explains the skin eruptions. That is why Hahnemann declared "it must be a matter of conscience for the physician . . . . to direct all his endeavours to cure, first of all, the internal malady, whereby the cutaneous eruption will at the same time be removed." As homœopaths, a knowledge of all that relates to the causation of disease gives us a clue according to the Law of Similars to that which may cure disease. Dr. Bach's conception that the non-lactose-fermenting bacilli in the bowel are closely related to the causation of the many manifestations of psora

Thomas Dishington

A. Statement summarising the homeopathic disease concept
Disease is not an entity to be expelled from the body, but a dynamic error in the life forces, an unbalance in the vital functions that can be corrected only by the recuperative power within the living cells themselves. - Dishington [13]

A[1] A reinterpretation and expansion
Disease is not an entity to be expelled from the body, but a [systems-disturbance] *that can be corrected only by* [engaging the intrinsic potential for self correction] *within the living* [systems] *themselves*, [and eliminating any extrinsic factors* which push the system towards chaos] - My brackets

* What Dishington refers to as the 'vicious cycle of [the] disease process' and which might also be termed *block to cure*.

B. Disturbed bacteria-host symbiosis is a systems disturbance
The bacteria cannot be looked upon as the sole causation of the disease, but are present because of the dynamic error within the life of the patient, and because they are closely related to that dynamic error they are within themselves the reflex of the host and they are the carriers of infection from the host. - Dishington [13]

B[1] The non-lactose fermenting populations in the stool of some ill patients are intrinsic to their systems-disturbance. The profile of the organism(s) present is specific to the illness state.

C. The autologous vaccine or nosode can deliver the systems-information required to correct the disturbance.
A nosode therefore made up from, say 100 non-lactose fermenting bacilli of one group ... contains the ... reflex of one hundred patients with that [systems-disturbance]. - Dishington [13]

C[1] Bach and Paterson supported the use of polyvalent nosodes derived from many individual cultures, from many patients. The strengths and weaknesses of this are discussed in the next section.

The Influence of the homeopathic remedy on bowel microecology

John & Elizabeth Paterson (& Dr Runcie - back)

Bach and Paterson demonstrated that the bacterial profile of the stool showed patterns of correspondence with the homeopathic remedy used. For example, if you give a patient homoeopathic Sulphur he/she will be observed to yield Morgan. [21]

John Paterson

Between 1927 and 1932, Paterson cultured 8000 stool specimens and he published some preliminary findings. [21] During this time Paterson observed: '*Homoeopathic potencies are capable of completely altering the bacterial flora of the bowel, and this fact has been demonstrated in many hundreds of cases.*' Paterson, J. *Potentized Drug and its Action on the Bowel Flora.*

After treatment with homoeopathic *Sulphur* or *Calcarea*, patients were found to have increased numbers of '*Morgan*' in the stool. Patients treated with *Lycopodium clavatum* yielded a '*Morgan type*' organism with a different fermentation profile, which was given the name *Morgan-gaertner*. [21]

'*Here, on a bacteriological basis, are found these three remedies in association under one main type, which however, can be subdivided. Into one subgroup comes sulphur and calcium, while in the other Lycopodium stands alone.*' [21]

This 'sub-typing' of *Morgan* (Bach) into Morgan pure and *Morgan gaertner*, arose as a result of remedy experimentation. (See fermentation profiles on page 1.22.)

Over the next few years 12.000 specimens were collected from medical attendees and analysed. Using the bacteriological methods of that time the six groups of non-lactose fermenting bacilli were isolated (table 7) and 'identified' in the stools of homeopathically treated patients.

Organism	%	%
B. Morgan pure	5.68	14.82
B. Morgan-gaertner	3.27	
B. Morgan "X"	2.73	
Unclassified 'hybrid'	3.14	
B. Proteus pure	2.27	3.37
B. Proteus hybrid	1.46	

table 7

Bacterial adherence

"Recent developments indicate that some adhesive bacteria are able to recruit a variety of structurally diverse host proteins, adhesive glycoproteins, growth factors and cytokines, by initially binding heparin and functionally similar sulphated polysaccharides to their surfaces, whence they serve as non-specific, secondary recruiting sites for other host molecules."

G.T.Macfarlane *Colonic ecosystem*

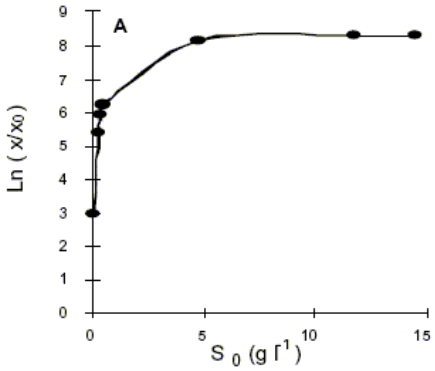
Why do remedies increase the stool numbers of certain organisms ?

Does homeopathic sulphur alter the bacterial ability to 'recruit' adhesive glycoproteins by altering their ability to bind sulphated polysaccharides? Do populations become less adherent and increase in the luminal content as a result? (Resulting in Paterson's observation of increased stool numbers.)

If homoeopathic remedies can alter the surface adherence of any population of bowel organism, or if it can alter any aspect of their gene expression, viz.enzyme function or substrate utilisation, there will be systems consequences.

PROPOSITION

If a homoeopathic remedy can alter the surface adherence of any population of bowel organism, or if it can alter any aspect of their gene expression, there will be changes in enzymal activity, substrate utilisation, leading to tertiary systems-effects.



Variation in *M. Morganii* bacterial concentration at different substrate concentrations

Here are some extracts from current reviews of bowel ecology:
[my underlining of some key points]

1. Metabolic activities of flora and enzyme expression

To get an idea about the role of a given organism in the human gastrointestinal tract it is necessary to know its metabolic capabilities. This requires either growing this organism in the laboratory and studying its metabolism in vitro or developing methods for the in situ-detection of bacterial activities.

Growing a microorganism in a medium may be a difficult task if the optimal growth conditions are not known and the organism under study does not grow on conventional media. However, even if growth conditions can be found, it is not quite clear whether the activities observed in vitro are also relevant for the in vivo situation.

One of the major tasks in future research will therefore be directed to the development of methods for monitoring enzyme expression and activity in situ at the cellular level.

Therefore, classical microbiological techniques and molecular based techniques are not mutually exclusive but complementary.

M. BLAUT *Assessment of bacteria in the gut microbial ecosystem*

2. Bacteria/bacteria interactions

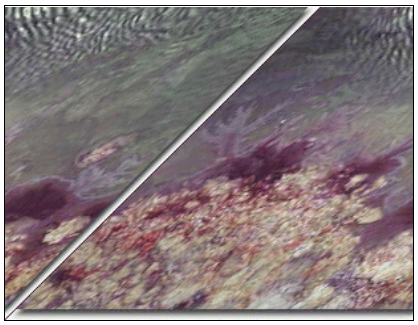
Major substrates of the intestinal flora are dietary constituents that escape digestion in the small intestine and endogenous sources such as mucins and proteins from sloughed cells and the host's digestive enzymes. The availability of substrates is a major factor for the development of bacterial communities. Interactions between bacterial population groups are largely based on cross feeding which means that one bacterial population group utilises certain degradation products formed by other bacteria as substrates. The variety of potential substrates formed during microbial breakdown of the primary substrates increases during the transformation of polymeric substrates to oligomers and monomers and other cleavage products.

M. BLAUT *Assessment of bacteria in the gut microbial ecosystem*

3. Multispecies communities - Biofilms

Other types of interactions between bacteria may involve the formation of antibacterial substances inhibitory to the growth of certain bacterial groups. In such a situation, the ecological niche opened by the inhibition of one group of bacteria may facilitate the establishment of another bacterial group which has the same substrate spectrum but is not susceptible to the antimicrobial compound. The ability of one species to survive in this habitat may depend on its ability to establish as a member of a multispecies community as present for example in so called biofilms. This in turn may depend on the presence of partner organisms required for attachment.

M. BLAUT *Assessment of bacteria in the gut microbial ecosystem*



Biofilm

4. Cell to Cell Signalling

The formation of multispecies assemblages as encountered in biofilms would be expected to require cell-cell communication to occur. Exchange of information between bacteria may be brought about by the formation of signal molecules that are released into the environment and can be sensed by other bacteria. Such signals may influence the behaviour and metabolic capabilities of bacteria by way of modulating gene expression.

In spite of the large number of bacteria and species present in the human intestine only very few examples of cell-cell signalling have been discovered. It can be assumed that most cases of cell-cell communication in this habitat have not yet been discovered.

M. BLAUT *Assessment of bacteria in the gut microbial ecosystem*

5. DNA transfer

Bacteria may not only influence other bacteria by the release of signal molecules, but also by the transfer of genetic material between bacteria. Although the principal mechanisms of DNA transfer are known it is not clear to which extent gene transfer occurs between bacteria in the gastrointestinal tract.

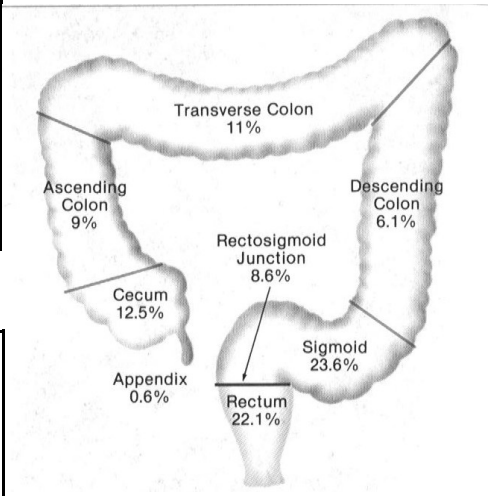
Gene transfer in the gastrointestinal is important because it has possible implications for the spread of antibiotic resistances and the ability of bacteria to adapt to environmental changes.

M. BLAUT *Assessment of bacteria in the gut microbial ecosystem*

6. Regional availability

The large intestinal microbiota is characteristically viewed as being a homogeneous entity, yet the proximal colon and distal bowel differ markedly in relation to their nutritional availabilities and physicochemical attributes.

G.T. Macfarlane Colonic ecosystem



7. Implications of non-culturable organisms

Except in very broad terms, little is known of the metabolic relationships that exist between individual bacterial communities in the colon, or of the ecology and multicellular organisation of the microbiota.

Moreover, a number of molecular studies have suggested that only a small fraction of bacterial species in natural communities, such as the gut, are culturable, thus, while we can readily determine that the ecosystem contains considerable numbers of phylogenetically and physiologically different bacteria, the relative population sizes and types of non-culturable organisms present in the microbiota are largely unknown.

G.T. Macfarlane Colonic ecosystem

8. Adherent vs. Non-adherent Populations

In the large gut, individual bacterial species and assemblages of microorganisms exist in a multiplicity of different microhabitats and metabolic niches, on the mucosa and in the mucus layer, as well as in the gut lumen. Examination of intestinal material by scanning electron microscopy and fluorescent light microscopy shows that most of the bacteria are not freely dispersed, but occur in clumps, and in aggregates attached to plant cell structures and other solids.

With respect to the numerically predominant culturable species, bacteria attached to surfaces in the gut lumen appear to be phylogenetically similar but physiologically distinct from non-adherent populations.

These adherent organisms are more directly involved in the breakdown of complex insoluble polymers than unattached bacteria, which provides a competitive advantage in the ecosystem. Close spatial relationships between bacterial cells growing on surfaces are important in other ways, particularly in relation to metabolic communication between different groups of microorganisms in the microbiota.
Furthermore, they are ecologically significant in that they minimise potential growth limiting effects on secondary cross-feeding populations, that are associated with mass transfer resistance...

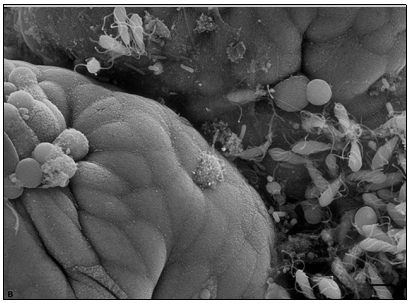
G.T. Macfarlane Colonic ecosystem

9. Relationship between mucosal populations and health

In healthy people, mucosal populations are more difficult to study than faecal bacteria due to difficulty in gaining access to the bowel, and has restricted studies on these communities.

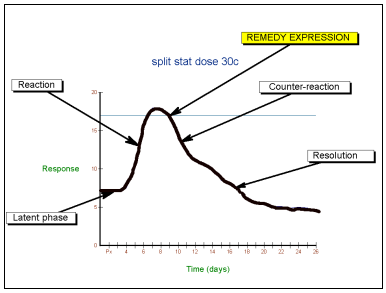
Consequently, little information is available concerning the composition, metabolism and health-related significance of bacteria growing at, or near the mucosal surface.

G.T. Macfarlane *Colonic ecosystem*



See also: S. Swift *Cell-cell communication* MEHD

Introduction to materia medica of the bowel nosodes.



Most experienced prescribers recognise that remedies prescribed on the principle of similars will often evoke some kind of response, even when the remedy is not optimally selected. This response may take the form of a proving symptom or a partial resolution or, perhaps, a short-term improvement. These reactions may indicate that you are on the right track (with a near or partial similimum*) and may suggest the use of a related remedy or a change in potency or frequency of dose.

Many nosodes, however, do not appear to evoke any response at all, unless they have some aetiological relevance in the case, or reflect a disturbed 'systems relationship' with an organism. It is common for beginners to be come discouraged by a few prescribing failures and then overlook the nosodes altogether.

The 'non-miasmatic' nosodes are almost universally prescribed on the basis of an aetiological model, relating to some kind of infective event (current, past or inherited). Infections involve host-organism relationships which are complex and dynamic.

Some infective agents can have a very long-term relationship with the host organism (TB, gonorrhoea, syphilis). These chronic infections generate a variety of immunological, physiological, metabolic and endocrine compensations in the host. Along with changes in the life-fabric of the sufferer, come idiosyncrasies of emotion and behaviour. The 'miasmatic' picture that emerges is highly differentiated for these remedies, in contrast to the almost non-existent remedy pictures for acute pathogens.

The patient who has a chronically dysbiotic bowel following infection, prolonged stress and antibiotics, will also develop some idiosyncrasies of response, various local pathologies and systemic disturbances. Miasmatic tendencies and vulnerabilities probably also emerge, just as they do following protracted gonorrhoea and TB.

Most true bowel pathogens (see *enterobacteraceae* in Section1) evoke acute symptoms (nausea, vomiting, diarrhoea), which are broadly similar, regardless of the pathogen. This lack of differentiation is unfortunate for the homoeopathic physician wishing to give the correct nosode. The sub-acute pictures (fatigue, disturbed bowel habit, bloating, flatus and secondary infection), are a little more differentiated.

The metabolic, endocrine and immunological disturbances of the chronic phase is often the most differentiated part of the clinical picture. It is this picture which is recognised and treated specifically by the homeopathic physician, or subject to suppression with steroids by the allopath.

Although the bowel nosodes clearly have a more detailed symptom picture than, say, *Staphylococcus*, their leading symptoms are not as characterised as *Medorrhinum* or *Tuberculinum*. However, they work as profoundly as any of the 'big' nosodes when correctly prescribed !

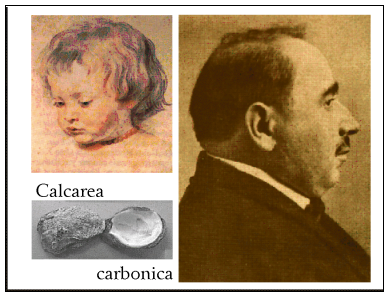
* Partial or truncated responses can also indicate various blocks to cure, including those for which Bowel Nosodes are indicated.

Nature and Origin of the Materia medica Data for the Bowel Nosodes

Infective and post-infective syndromes are qualitatively different from the symptom complexes that emerge in proving experiments. Bowel nosodes have been used empirically for many years and we can distinguish three broad categories of data within their 'symptom pictures':

~ Hints relating to **predisposed types**: ie. the kinds of people who are more likely to develop a dysfunctional relationship (dysbiosis) with that class of organism, or who are predisposed to chronic illness after exposure.

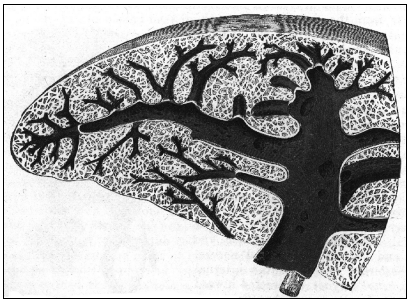
C This cross-matches loosely to some of the known constitutional pictures.
(Mind & Typology)



NB. Conversely, dysbiotic patients have been shown to externalise specific organisms after taking the related constitutional remedy. (eg. Morgan pure has been isolated in increased numbers from the stool of dysbiotic Sulphur-sensitive patients, following administration of homeopathic Sulphur potencies.)

~ Hints relating to the **points of vulnerability** that emerge with different kinds of dysbiosis: ie A locus of secondary infection, inflammation or disordered function.

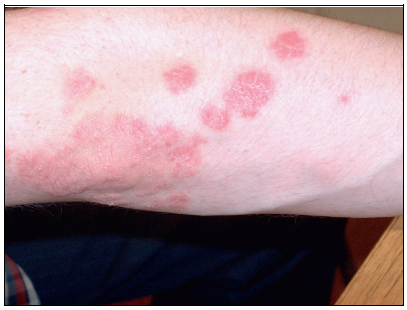
C This cross-matches with the known organ affinities of certain classical remedies. (Locals)



Symptomatic comparison with key gastro-intestinal remedies may be particularly important in the management of inflammatory bowel disease.

~ Information on the primary and secondary **consequences of living with a certain kind of dysbiosis.** (physiological, immunological, endocrine decompensation)

C This cross-matches to the symptom-pictures of classical remedies.(Generals, Fever, Chill, Modalities of local symptoms)



This keynote information has been summarised in a table for each nosode in the following materia medica section. (These tables are my interpretations of the available data. The emphases and suggested remedy relationships may differ from some of the existing literature.)

No. 7

BACILLUS NO.7 (PATERSON)

Citrobacter spp. (? *C. koseri*), (+/- *Enterobacter cloacae*)

Hypotheses	Features		Similars
Predisposed types	Tense overworking types ? Dark, pale colouration		Kali salts
Main aetiology	Infective		
Secondary aetiologies	Overwork Over-indulgence Drugs and antibiotics		
Emergent disturbance	Systemic		
Secondary disturbances	Fatigue states Inflammatory arthritis		
Principle locations	Systemic	**FATIGUE**	
Secondary locations	Neuromuscular Joints	Cracking joints	Bryonia
GI pathophysiology	Diffuse		Carbo veg.

The keynote for the use of this nosode is extreme mental and physical fatigue. It has developed insidiously and is often associated with wasting of muscle groups, perhaps with myocardial weakness which may strike suddenly. Its site of action is the neuromuscular junction.

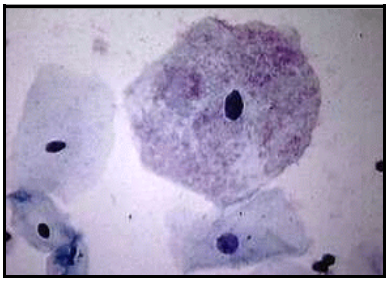
Appearance: Dark, pale; puffy.
Mind: Tense. Tired. The thought of doing anything is sufficient to exhaust.
Face: +/- Angio_neurotic oedema:
Respiration: +/- Asthma; bronchial catarrh; tough sticky mucus, difficult to raise; worse at 2 A.M. Here Kali carb is the associated remedy.
Neck and Back: Stiff neck: 'cracks like a nut.'Fibrositis neck and shoulders: Fibrous rheumatism of neck and back, abdominal muscles; shoulders and arms. Spinal osteoarthritis. Backache > heat and rest. < damp and cold and on commencing to move. Spondylo-arthritis with ankylosis.
Circulation: Slow pulse rate, often with lowered blood pressure : Faintness on standing long and after sudden exertion. Excessive perspiration.
Stomach: Eructation. No vomiting.
Abdomen: Pain in liver region. Flatulence.
Digestive System: All the symptoms can be related to general lack of nerve & muscle tone; sense of fullness after food; flatulence/distension of stomach
Upper limbs: Rheumatoid arthritis shoulders; elbow, wrists. Ganglion right hand. Fingers swell. Blood vessels burst in fingers. Rheumatism wrist, thumb osteoporosis.Wrists and ankles immobile. Rheumatoid arthritis.
Lower limbs: Joints swollen and painful. Fibrous rheumatism thighs. Limbs stiff. Pain shoots up and down leg. Stabbing pain hip. Cramps in leg at night. Osteoarthritis knees. Periarthritis knees. Left hip fixed, arthritis.
Rheumatoid arthritis, knees and ankles. Feet painful. Gout in left toe.
Neuromuscular System: Relaxed fibrous tissue,with tendency to the formation of rheumatic nodules. Backache, cannot stand long without
Genito urinary System: Feeble urinary flow; loss of sexual function; premature senility. Pain in vulvae, no leucorrhoea.
Bowels: Constipation. Haemorrhoids.
Skin: Cracks knuckles_tips of fingers. Cracks palms.
Circinate eruption hands (palms) hot, nippy and scaly. Paronychia.

BACILLUS No.10 (PATERSON)

?? *Gardnerella*

No.10

Hypotheses	Features		Similars
Predisposed types	Active, ? instinctual people ? Sexual ? Relationship issues		*Sepia* 'Sycotics' *Medorrhinum*
Main aetiology	Opportunistic infection ? Sexually transmitted diseases		
Secondary aetiologies	? Overuse of herbals and homeopathy ? Dietary fads		(Progesterone)
Emergent disturbance	Disturbances of surface immunity Persistent inflammation -**mucus membranes**		
Secondary disturbances	Systemic 'toxicity' with fatigue and loss of wellbeing		
Principle locations	Mouth, **VAGINA**	Inflammatory	*Borax*
Secondary locations	Anus Urethra Gallblader		*Nit-ac* *Hydrastis*
GI patho-physiology	Indistinct dysbiosis.	Disturbed bowel habit	



Marker squamous cell appears darker with adherent G - ve Gardnerella

This was the tenth non-lactose fermenting type of bacillus 'isolated' in the laboratory by Paterson.

Appearance: Fair; florid. Dark; pale
Mind: Anxious; active; irritable; depressed
Head: Headache forehead, left eye.
Nose: Catarrh. Mouth: Spongy gums. Halitosis
Chest: Asthma. Cough < in the morning; sputum difficult. Panniculitis chest wall. Lipoma lower ribs
Desires & Aversion: Averse: egg; bread; tomato; tea.
Averse breakfast. Craves: sweets; chocolates: fried fish.
Upset by : egg and fat. Anorexia.
Stomach: Nausea; vomiting.
Abdomen: Occasional pain gall bladder.
Body and limbs: Tenderness coccyx. Rheumatism of thigh.
Rheumatoid arthritis left knee. Paronychia.
Urinary: Frequent micturition.
Genitalia: Pruritis valvae. Leucorrhoea fishy odour; greenish; corrosive. Flesh groin raw; dry and cracked. Urethral caruncle.
Bowel: Bowel motion first thing in the morning. Pain LIF, RIF
Bowel motion sluggish; very often diarrhoea. Pruritis ani.
Skin: Warts on hands_numerous flat or pointed (after working with pickled hams). Dermatitis flexures ? allergy to drug given for asthma. Ringworm.
Perspiration in axilla.

Dys.

DYSENTERY CO. (Bach) *Shigella* spp. (+/- ?? *Salmonella* spp.)

Hypotheses	Features		Similars
Predisposed types	Unrelaxed. Trim and health-conscious but overworked and overconscientious. Fine complexioned but showing signs of strain. Perhaps driven by self-doubt and insecurity. Anticipates misfortune.		*Arsenicum* & salts *Argentum nitricum* ? other nitrates
Main aetiology	Drugs		*China officinalis* *Chininum* salts
Secondary aetiologies	Infective / antibiotics or both		
Emergent disturbance	Endocrine disturbances Thermoregulatory disturbances		*Thyroidinum*
Secondary disturbances	Immune hypersensitivity Hayfever & rhinitis		*Tuberculinum* *Carcinosinum*
Principle locations	Endocrine and systemic	**Thyroid** dysfunction **FATIGUE** states	*China officinalis* *China ars.* *Elaterium* *Gelsemium* *Parthenium* *Veratrum album*
Secondary locations	Mucous membranes Endothelium Respiratory tract Cardiovascular	Secondary infective and low grade inflammatory processes. Local dysbiosis Chest pains - (? mainly functional or psychosomatic)	*Ammonium carb* *Bacillinum* *Pulsatilla* *Cactus grandiflora* *Digitalis purpurea* *Spongia tosta* *Tuberculinum bov.*
GI pathophysiology	All levels of GI tract but especially **pylorus** and duodenum	Indigesion Diarrhoea	*Abies nigra* *Abies canadensis* *Ptelea* Veratrum album

Suggested reading for Dys co: *The Pathogenesis of Dysentery* by Thomas Dishington

Fæcalis

FAECALIS ?Bacillus Faecalis Alcilagenes

Hypotheses	Features		Similars
Predisposed types	Anger with people who are trying to help. $^{2}/_{3}$ **Stress** through inability to communicate. $^{2}/_{3}$ Over-exposure to **hospital** environments. $^{3}/_{3}$		*Sepia* (Kali-m) (Bar-c) (Ant-c)
Main aetiology	Social stress $^{2}/_{3}$ Communication issues / frustration $^{2}/_{3}$ **Poor diet** (incl. hospital food / junk food) $^{3}/_{3}$		
Secondary aetiologies	? Cancer $^{1}/_{3}$ / $^{2}/_{3}$? Surgery $^{2}/_{3}$? Chemotherapy / radiotherapy $^{1}/_{3}$? Inherited dysbiosis $^{1}/_{3}$? Birth trauma & post operative stress $^{1}/_{3}$		*Carcinosinum* *Condurango* *Hydrastis*
Emergent disturbance	Post operative diarrhoea / constipation abdominal distension		*(Croton tig.)* *(Opium)*
Secondary disturbances	Swelling / lymphadenopathy		*Apis* *Vipera*
Principle locations	Large bowel $^{3}/_{3}$ Interstituum Lymphatics		*Viscum album* *Vinca minor* *Taxus*
Secondary locations	(Skin) (Urinary tract)		
GI pathophysiology	Alternating stool $^{2}/_{3}$ << constipated phase		

Gært.

GAERTNER (BACH) - (*Salmonella* spp.)

Hypotheses	Features		Similars
Predisposed types	Bright active types. Often intellegent. Often fair hair; blue eyes; freckles. Thin, pale, nervy people.		*Phosphorus* *Silica* *Merc vivus* *Tuberculinum*
Main aetiology	Infective		
Secondary aetiologies	Antibiotics, drugs or both		
Emergent disturbance	Immune or autoimmune		*Carcinosinum*
Secondary disturbances	Inflammatory arthritis Inflammatory bowel disease Malabsorbtion syndromes		*Sanicula aqua* *Phosphorus salts* *Silicates*
Principle locations	Small intestine	Malabsorbtion	
Secondary locations	Lymphatics Glandular Spleen Endothelium Synovium		*Asafoetida* *Ceanothus*
GI pathophysiology	Post-salmonella dysbiosis.	Diarrhoea Bloody stool Defective absorption	*Aloe* *Podophyllin*

"*In all varieties of neurasthenia, I know of no single remedy to compare with the Gaertner nosode.*"

(C.E. Wheeler)

The keynote of Gaertner Co. is "nutrition" and this nosode might suitably be named as 'the children nosode', because in clinical picture you will find something of practically all the nutritional disorders so common in the child, but it is equally found useful in the other extremes of life associated with malignancy. Marked emaciation may be taken as an indication for the use of this nosode.

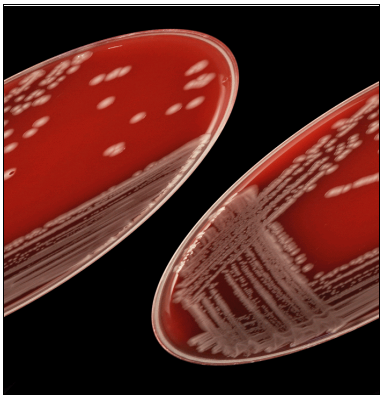
(Aggrawal - after Elizabeth Paterson)

Gaertner has great relation with malignancy. Dr. John Roppe, the pathologist found that Gaertner bacillus was in the stool of malignant cases.

'*Children are hypersensitive to all impressions, psychical or physical; overactive brain with under nourised body.*'

(John Paterson)

Gaertner - continued



Eyes: White sclerotics. Styes.

Ears: "*For cases of children who get a discharging ear every time they cut a tooth, give one dose of Gaertner 30, followed the next day by the dose of Silicea 30, and repeat the Gaertner as soon as there is any sign of a further out break of this ear discharge*." (Beta)

Nose: Polypi. Catarrh.

Mouth:
Salivation. Herpes. Dry scaly eruption. Teeth black. Deep fissures in tongue.

Neck and back: Pain in hip and back (severe).

Desires and Aversions:
Craves: oatmeal (porridge and oatcakes); cheese; eggs; milk pudding; sugar and sweets.
Averse: bread; butter; butcher meat; fish.

Stomach: Pain in stomach. Vomits everything: vomiting < after sweets. Headache and vomiting : acidosis attacks. Dilated stomach. Stomach attacks or gastro-intestinal attacks.

Digestive System: B. Gaertner has its greatest action on digestive tract. *Children fed on artificial food have poor digestion*.
The inability to digest fat - coeliac disease; ketosis, "*intestinal infantilism*" are disease complexes found under the 'provings' of this nosode.

Extremities:
Chilblains hands in winter. Bites nails. Intractable rheumatic cases. (Laura Hurd).

Urine: Blood and mucus with urine. Burning in urethra.

Genitalia: Profuse offensive leucorrhoea. Hydrocele. Pruritis vulvae.

Bowels:
Constipation. Diarrhoea - offensive : attacks every few weeks.
Chronic gastroenteritis; tabes mesenterica. Blood and mucus in stool.

Sleep: Sleep walking. Restless sleep; night terrors.

Skin: Urticaria; heat spots. Boils arms and legs.

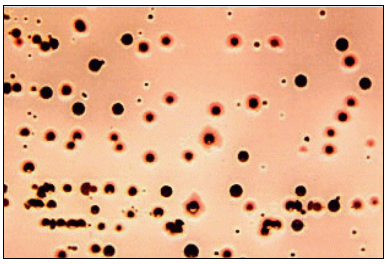
Eruption: Back, head and neck. Circinate eruptions on sternum.

Perspiration < at night.

MORGAN - GAERTNER (PATERSON)

(*? Aeromonas, ? Edwardsiella tarda, Escherischia blattae, Haffnia alvei*)
One of two discreet isolates from Morgan (Bach)

Morg-g.



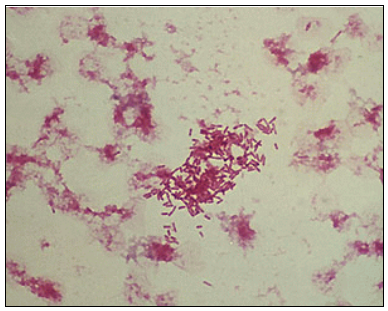
Hypotheses	Features		Similars
Predisposed types	Mainly pale, apprehensive anxious types Perhaps socially dysfunctional. Gastrointestinal vulnerabilities.		***Lycopodium*** *Chelidonium*
Main aetiology	? Sedentary habit , ? poor diet		
Secondary aetiologies	Drugs, Food-bourne bacterial toxins Stress		*Nux vomica* *Sulphur*
Emergent disturbance	Low grade chronic GI and GU inflammation		*Chenopodium* *Taraxicum*
Secondary disturbances	General immunological ? Uterine hyperplasia / **menopause**		*Pulsatilla* *Lachesis* *Sepia* *Sanguinaria*
Principle locations	Colon	Altered bowel habit **flatulence**	
Secondary locations	Liver & **biliary** Renal tract	Biliousness / nausea Stones / renal colic ? chronic cholangitis	*Copiava* *Sarsasparilla* *Calcarea renalis*
GI pathophysiology	Flatulence	Distention	*Carbo vegetabilis* *Dichepetalum*

Morgan Gaertner has strong emphasis on the urinary tract; renal stones, renal colic, cystitis, vulvo-vaginitis. Flatulence is a common symptom.

Mind - Irritable; quick tempered; impatient. Tense; nervous. Restless; weepy; depressed. Jealous; particular; apprehensive. Fears crowds; excitement & company.
Head: Congestive headache. Flushed face.
Eyes: Blepharitis. Styes. Cysts on lids.
Ears: Otitis. Mastoiditis. Boils in ears. Singing in ears.
Nose: Nasal catarrh + +. Post nasal catarrh + +. Dry catarrh- crusts, ulcers in nose. Polypus nose. Red nose. Herpes nose. Epistaxis. Sinus infection.
Mouth: Bitter taste +. Bad taste. Gums inflamed.
Pyorrhoea. Tongue burning. Pins and needles sensation in tongue. Tongue glutty in morning; saliva glutty. Dirty tongue. Fissures at angles of mouth.
Stomach: Flatulent indigestion: eructation excessive.
Eructation of bad odour. Sour mouthfuls (Pyrosis). Fullness epigastrium, unrelated to food. Pain in epigastrium after food. Vomiting after food -afternoon or night. History of duodenal ulcer.
Abdomen: Distended feeling. Distended colon. Pain in right and left hypochondrium and epigastrium. Pain right and left iliac fossae. Pain in ileocoecal region. Pain gall bladder: cholecystitis. Tenderness of gall bladder. Pain in right and left shoulder blades.
Extremities: inflammatory arthritis
Urine: Enuresis. Cystitis. Renal colic, renal stone. Nephritis; pyelitis.
Genitalia: PMT Dysmenorrhoea, Pruritis Leucorrhoea - heavy; brown; bad odour.
Bowels: Flatulence excessive in bowel. Constipation more than looseness. Sluggish bowel. Looseness urgent. Stool _ hard, dry with mucus.
Anus: Piles, painful, itchy bleeding. Anal fissure; pruritis ani. Prolapse rectum. Mucus per rectum even if motion is not stiff.
Sleep: Insomnia (common). Drowsy after food. Night terrors, shouts in sleep.
Skin: Psoriasis - elbows; knees and ankles, legs, or body, even toe nails. Eruption on thighs; wrist where metal contact. Herpetic eruption on soles of foot.

MORGAN PURE (PATERSON) (? Morganella morganii) (+/- other species)

Morg-p.



Hypotheses	Features		Similars
Predisposed types	Heavy, **plethoric** Sluggish		**Carbons** **SULPHUR**
Main aetiology	Consumption of *Morganella* infected food /Chronically poor diet.		*Hepar sulph.* Sulphur salts
Secondary aetiologies	? Drugs, steroids, infection, alcohol Weaning - introducing solids		*Rhus tox.* *Thuja* *Tuberculinum*
Emergent disturbance	Physiological / histaminogenic ? centred around problems of elimination		
Secondary disturbances	Metabolic Circulatory		**HISTAMINE**
Principle locations	**Skin** Circulation		*Sulphur* *Graphites* Skin remedies
Secondary locations	All systems / Joints Airways		
GI pathophysiology	Constipation Haemorrhoids Diverticular disease		*Sulphur* *Hamamelis* Carbons

Morganella morganii SYNTHESISES HISTAMINE

Appearance: Florid: dark more than fair. Pale: either dark or fair. Red lips, red cheeks, a florid type of countenance.
The build is portly and appearance plethoric, possibly with blueness of extremities
Mind: Tense; active; weepy; depressed; irritable. Introspective, anxious and apprehensive about state of health. Avoids company.
Head: Congestive headaches, with flushed face.
Headaches: frontal, occipital and headache on vertex. Migraine.
Scalp: Scalp sensitive. Hair falling out. Complete alopecia.
Face: Acne rosacea. Hairy face.
Eyes: Lids granular. Conjuctivitis. Tarsal cysts. Styes. Iritis; keratitis; phlyctena.
Ears: Catarrhal deafness. Otorrhoea. Noises ears. Meniere's disease.
Nose: Catarrh. Post nasal catarrh. Sinus and antral infection. Dry mucous membrane. Cracks angle nose : crack nose. Sense of smell lost. Epistaxis.
Mouth: Mouth and lips dry_lips stiff in the morning. Ulcers in the mouth. Salivation. Angles at mouth cracked; lips very red. Bad taste in the mouth. Burning in tongue; tongue raw and dry, coated slimy, swollen.
Throat: Thyroid enlarged. Throat dry and burning.
Throat parched, row and granular. Recurring tonsillitis: cheesy pieces come out. Pharyngitis; laryngitis; tracheitis. Apple core sensation: easy choking.
Respiration: Bronchitis each winter. History of pneumonia or broncho-pneumonia; never well since. Bronchitis and asthma. Emphysema. Suffocative attacks at night. Shortness of breath. Morning cough; dry tickling cough -loose or sticky. Infantile asthma. This is also a remedy of value in chest infection where the indicated remedy fails to act or in cleaning up residual infection after a chest infection, especially in cases following Sulphur or Natrum Sulph.
Neck and Back: Pain in neck. Fibrositis neck and shoulders. Pain generally < night < heat: > moving: < on beginning to move. Arthritis: spine, sacro_iliac joints.
Chest: Fibrositis chest wall. Emphysema. Angina. Pain since shingles.
Desires and Aversions: Fond of fats: sweets: eggs and butter, more than averse. Upset by fats and eggs: or avoids fats and eggs.

MORGAN PURE continued

Stomach: Waterbrash: heartburn, Sour, acid, bitter mouthfuls. Eructations; pyrosis.
Burning in throat and stomach. Pain and acid > with food. Nausea, vomiting haematemesis Duodenal and peptic ulcer. Bilious attacks.
Abdomen: Epigastric pain or discomfort.
Pain right and left hypochondrium / iliac fossae.
Pain in liver and gallstones. 'Epidemic jaundice' in children (Griggs) Flatus.
Redness and moisture at umbilicus. Bad odour.

Histamine producing bacteria isolated from *Trachurus murphyii.*

Organism	log CFU	Histamine ($\mu g\ ml^{-1}$)
Morganella morganii	9.51	144.0
Proteus vulgaris	9.79	26.41
Aeromonas spp.	8.08	5.24
Pseudomonas spp.	6.23	6.44
Pseudomonas putrefasciens	5.10	2.45

Digestive System: Congestion. Heartburn and dirty tongue. Congestion of liver. Bilious attacks with severe headache especially at menopause.
Circulation: Varicose veins. Phlebitis. Flushes. Varicose ulcer. High blood pressure. Sluggish circulation. Cerebral thrombosis. Easy bruising.
Tendency to haemorrhoids.
Upper limbs: Shoulder pains ++ rheumatic shoulders. Pain in arms; wrists, hands. Pain keeps awake at night. Hands too hot at night. Fingers stiff in morning. Rheumatoid arthritis wrists Rheumatism thumb: osteoporosis.
Lower limbs: Tingling legs feet numb. Growing pains. Coldness in patches. Knee swollen: grating. Periarthritis knee. Knee joint stiff. Osteoarthritis knee. Pain in soles of feet. Metatarsophalangeal joints painful and swollen. Pain in the heels. Thick skin soles of feet with cracks heels. Feet too hot nocte: offensive foot sweat. In female adolescents chilblains of feet and toes.
Urine: Cystitis : with frequency and pain. Urine of strong smell. Micturition is often burning and vulvovaginitis may be present. Enuresis. Urine sugar.
Genitalia: Pruritis vulvae and vagina + + +. Menorrhagia and metrorrhagia Polypi and fibroids of uterus. Leucorrhoea : corrosive, offensive; brown; green; yellow. Boils vulva. Urethral caruncle; bartholinitis. Dyspareunia.
The congestive headache with menstrual onset +/- ovarian pain (congestive dysmenorrhoea) Congestive flushings of the menopause period.
Bowels: Constipation. Pruritis ani + + +. Piles bleeding itching or painful. Anal fissures. Bowel motion without help; loose; urgent in the morning.
Stool: May be pasty, foul and contain blood and mucus.
Skin: Hyperkeratosis and congestions of skin with fissuring. Itching eruption, worse from heat. Callosities. Perspiration profuse in axillae. Raw, red eczema exuding freely with itch and heat, bleeding from scratching.

Dr. William B. Griggs says, "*Morgan Bacillus has been one of my most successful remedies in eczema of young children*."

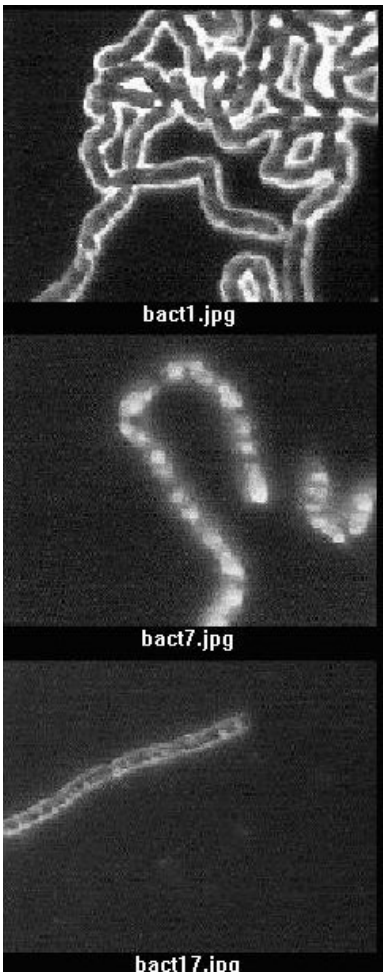
"*If any of you have a case of infantile eczema that is quite resistant, do not forget the Morgen nosode, it has cleared it many times.*" (T.K. Moore)

"*You take the moist type of eczema where the child scratches and has the exudate on its face and bleeds. I have seen Morgan clears that up after failure of Graphites, Psorinum and Medorrhinum. "The type of eruption which characterise this can be ascertained from a study of the provings of well known skin remedies found among the list of remedies associated with the Bacillus Morgan e.g., Sulphur, Graphites, Petroleum. There are few eczemas of the infants at the teething stage or later life, which do not require a dose of this nosode*." (Y. R. Agrawal)

Mutabile. ? Salmonella subspecies, ? E. Coli subspecies, ? Morganella

Mutabile.

Hypotheses	Features	Similars
Predisposed types	Unstable or changeable types Vacillating, Capricious	*Pulsatilla* *Cimicifuga*
Main aetiology	? Emotional stress	*Camphora*
Secondary aetiologies	Xenobiotics, Herbals and over-treatment with symptomatic drugs ? Poor, multiple homeopathic remedies ? Abuse of laxatives ?	
Emergent disturbance	Mood swings Feelings of insecurity	*Asarum europeum* *Medorrhinum*
Secondary disturbances	Urinary tract infection Diverticular disease UTI, asthma, eczema, perspiration	*Copiava*
GI pathophysiology	? Diverticulitis	*Lactuca virosa*

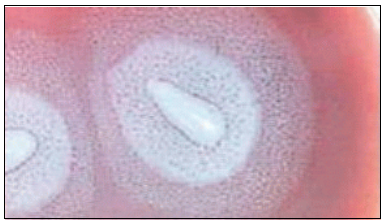


Rubrics from Complete Repertory v 3.0

MIND; COMPANY; desire for: mut [12]
MIND; IRRITABILITY: mut [12]
MIND; WEEPING, tearful mood; easily: mut [12]
FACE; SWELLING; eyes; around: mut [12]
STOMACH; VOMITING; blood: mut [12]
RECTUM; CONSTIPATION: mut [12]
BLADDER; INFLAMMATION; chronic: mut [12]
URINE; ALBUMINOUS: mut [12]
URINE; ODOR; beans, like boiled: mut [12]
RESPIRATION; ASTHMATIC: mut [12]
RESPIRATION; ASTHMATIC; alternating with; eruptions: mut [12]
BACK; PAIN; general; sitting, while; amel.: mut [12]
BACK; PAIN; general; standing; while: mut [12]
BACK; PAIN; general; Lumbar region, lumbago: mut [12]
BACK; PAIN; general; Lumbar region, lumbago; sitting; amel.: mut [12]
BACK; PAIN; general; Lumbar region, lumbago; standing: mut [12]
BACK; PAIN; general; Lumbar region, lumbago; standing; erect, impossible: mut [12]
EXTREMITIES; ARTHRITIC nodosities; Finger joints: mut [12]
EXTREMITIES; DISCOLORATION; Hand; blueness: mut [12]
EXTREMITIES; DISCOLORATION; Foot; blueness: mut [12]
SLEEP; FALLING ASLEEP; perspiration; during: mut [12]
SLEEP; RESTLESS: mut [12]
PERSPIRATION; NIGHT: mut [12]
PERSPIRATION; NIGHT; sleep, during: mut [12]
PERSPIRATION; SLEEP; during: mut [12]
SKIN; ERUPTIONS; alternating with; respiratory symptoms: mut [12]
SKIN; ERUPTIONS; eczema: mut [12]
GENERALITIES; ALTERNATING states: mut [12]
GENERALITIES; CHANGE; symptoms, constant change of: mut [12]
GENERALITIES; WEAKNESS, enervation; exertion, from; slight: mut [12]

PROTEUS (Bach)

Prot.



Hypothesis	Features		Similars
Predisposed types	Angry, **unstable** types Highly strung perhaps hysterical **Unrelaxed** **Autonomically aroused** Perhaps mainly thin and dark haired Perhaps stubbornly self-righteous and indignant. Unwillingness to accept change.		*Colocynthis* *Nux vomica* *Magnesiums* *Muriates* *Nitrates* *Natrum mur* *Staphysagria*
Main aetiology	Stress (especially persistent unremitting)		
Secondary aetiologies	Infective / antibiotics or both		*Helicobacter* *Sarracenia*
Emergent disturbance	Psycho-neuro-endocrine		
Secondary disturbances	Degenerative change at sympathetically innervated sites. Fibrositis at sites of chronic musculoskeletal tension.		*Cuprum met.*
Principle locations	Autonomically innervated systems and organs: - increased sympathetic activity	Circulatory including Raynaud's phenomenon	*Adrenaline* *Noradrenaline*
Secondary locations	CNS / PNS Musculoskeletal Cardiac	Stress headaches Fibrosistis	*Ignatia* *Natrum mur*
GI pathophysiology	Duodenal ulcer Irritable bowel		

Mind: Tense; irritable; depressed Outburst of temper, especially if opposed; will throw, kick or strike. Violence, spasms, particularly in the young.
Head: Headache frontal with sense of weight. Headache < before menses, < morning. Migrainous headaches are often with blurred vision. Meniere's disease.
Vision: Blurred vision during migraine. Burning pains. Acute pains. Pains > by pressure. Photosensitivity.
Nose: Impression of greasy discharge at the back of nose. Tightness.
Mouth: Angular cheilitis. Gums tender. Buccal ulcers. Taste salty.
Neck and Back: Fibrositis head and neck. Backache. Slipped disc.
Chest: Pain < with cold. < with exertion. Tightness; short of breath. Panniculitis.
Heart: Anginal attacks due to spasm of coronary capillaries.
Heart strain: E.C.G. coronary insufficiency, but no infarction.
Fond of: fats; sweets; salt; butter; eggs. Upset by: eggs.
Stomach: Acidity; heartburn; sourness. Flatulence. Hunger pain not relieved by eating. Nausea and migraine after meals. Frequent hiccough after meals. Vomiting after meals. Dilated stomach. Bilious at menstrual period.
Abdomen: Flatulence. Pain R.I.F.; pain L.I.F.
Upper Limbs: Contraction palms/fingers. Loss of grip strength. Hands numb in am.
Lower Limbs: Intermittent claudication. Cramps legs. Numb feet. Feet feel frozen. < cold atmosphere. Sciatica. Raynaud's disease.
Urine: Urethritis. Loin pain. Urine cloudy and foetid. Whitish fibres in the urine.

PROTEUS continued

Genitalia: Leucorrhoea profuse and offensive.
Leucorrhoea brown; thick; scalding. Vaginitis. Boil in vagina. Pruritis vulvae.
Blood streaked, brownish discharge before menses; menses with blood clots. Fibrous blood clots at the end of menses. Menses irregular, with blood clots for 7 days.

Bowel: Alternative diarrhoea and constipation. Emotional diarrhoea. Diarrhoea with headache and furred tongue. Constipation with empty urging.
Stools yellow, soft in morning after breakfast. Oxyuris. Sensation of ball in bowel.

Anus: Piles itch and bleed. Itch very bad. The child may suffer recurring rectal spasm.

Skin: Angioneurotic oedema, (cf Apis Mellifica) is found in the clinical proving of Proteus, also herpetiform eruption at the muco-cutaneous margins. Skin is light, sensitive and may be pigmented or the opposite, leucoderma; liable to flexure dermatitis, of knee and elbow. Herpes, hives and intractable dermatitis, usually pruriginous. Dermatitis back of hand with discharge (very bad). Pruritis intense. Anal and genital pruritis. Boils axilla. Erythema with papulopustular eruption - dry, scaly, crusting, itching; six months duration on chin and upper lip.

Proteus mirabilis is a histamine producing organism
"*Proteus can alter the soil in which psoric symptoms grow*" (A.C. Gordon Ross)

Perspiration under the arms, hands clammy. Copious perspiration from the axillae, falling in large drops. Nails split. Hair falls out.

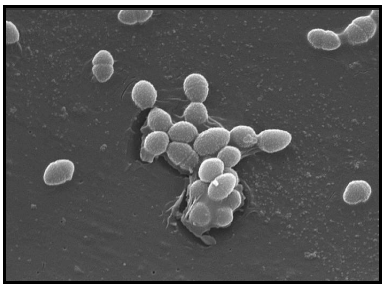
"*Suddenness of onset is outstanding characteristic of this nosode and this is seen in the herpetic eruptions of lips, mouth and eyes where pain is out of proportion to the size of lesion. Similarly if boils are present, they are equisitely tender, indurated, discharge little and are slow to heal. There is marked sensitivity to exposure to ultra violet rays.*" (Agrawal)

"*Convulsive and epileptiform seizures and meningismus in children during ferbile attacks often respond to the action of the nosode Proteus. Among the remedies related to Proteus are many chlorides especially Natrum Mur. Paterson noted an increase in Proteus after the last war and associated it with nerve strain, continued over a long period.*" (Agrawal)

Modalities: < in the morning on waking, from exertion, from climbing stairs, in stormy weather, heat, exposure to the sun and also in the winter, from cold.
Nightly aggravations and < from lying down. > from moderate temperature, from resting and in the mountains.

SYCOTIC CO. (PATERSON) (*enterococcus* +/-, ? *branhamalis catarrhalis*)

Syc.



Hypothesis	Features		Similars
Predisposed types	Overweight, but poor quality diet Sallow; Pale; anaemic. Puffy; greasy skin. Probably dark haired more than fair haired.		*Calc carb.* *Calc phos.* *Thuja* *Medorrhinum*
Main aetiology	Infective including viral URTI / measles and enterovirus infection		
Secondary aetiologies	immunisations, antibiotics, drugs, stress		*Morbillinum*
Emergent disturbance	Disturbed surface immunity		
Secondary disturbances	Catarrhal states, proliferative conditions		
Principle locations	Respiratory mucosae Sinuses, nasopharynx		*Bacillinum*
Secondary locations	Body orifices Skin GI and genital tracts Synovium	Otorrhoea Vulvovaginitis Dermatitis Arthritis	*Borax* *Thuja* *Sabina*
GI pathophysiology	Chronic irritation of whole alimentary tract; Distended. Children may have digestive difficulties with a history of enterovirus infection.	Nausea. Anorexia. Eructation bilious attacks. Pain and distension in epigastrium. Flatulence. R.I.F. Pain L.I.F.	

Mentals: Nervous; tense. Cross; restless; weepy; depressed; shy; sensitive; fussy. Exhausted. Mostly cold sensitive. Bites nails. Nervous irritability; quick temper; outbursts of temper from resentment. Fears of dark and of being left alone; fear of animals and dogs. This nosode is very rich in mental symptoms, which are very similar to *Gaertner*. The element of fear is outstanding, but there is additional factor of irritation. This may be manifested by outbursts of temper, suggestive of remedy Lycopodium, which is often complementary to this nosode.
The sycotic patient is always anaemic looking, never carries much colour in the face. Although the mentals of *Gaertner* and *Sycotic Co.* are similar, the physical appearance of the child is different. In contrast to the thin, undernourished child in *Gaertner*, the Sycotic child is often fat and flabby. Head sweat during sleep, chiefly from 12 to 4 am (cf. *Calcarea carbonica)*

"Calcium carbonate does not entirely cover the picture of the Sycotic child .....but here calcium bears the same relation to Sycotic co. as carbon does to Morgan co." (William B. Griggs)

Head: Irritation of meninges", sub_acute or chronic; headache from infection of sinuses; persistent headache particularly in a child _ which may be the prodromal sign of a tubercular meningitis (cf. Helleborus). Headaches are chronic, deep seated in nature, meningeal or sinusal origin. Headache weekly; every sunday morning. Headache < left side (lasts weeks); throbbing in heat and rest, in noise. Frontal congestive headache which lasts for many days. Sick headache < at menstrual period (before or after). Sweating of head at night. Headache slight, but recurring in child.
Scalp: Alopecia. Painful dry scaly spots. Premature grey hair.
Face: Puffy in morning especially under eyes. Acne rosacea. Erythema. Vesicular eruption on cheeks. Facial neuralgia. Hair on face and upper lip. Facial twitching .

SYCOTIC CO. continued

Eyes: Conjunctivitis. Tarsal cysts. Photophobia, Vitreous opacities.
Ears: Deafness. Otorrhoea. Excessive wax. Itching of meatus. Cracks under ears.
Nose: Nasal catarrh. Post nasal catarrh. Turbinates congested, sinus infection. Nose dry; crusting and burning. Polypi. Cracks in angles of nose Epistaxis. Sense of smell lost. Hay fever. Vasomotor rhinorrhoea. *Sycotic Co. is a remedy of great value in catarrhal conditions and violent cough.* (T.D. Ross)
Mouth: Lips dry/cracked. Angular cheilitis. Persistent herpes. Tongue sore; scalded. dry; fissured; furred. Deep ulcers on tongue. Bad taste; sense of taste lost. Paraesthesiae tongue. Ulcers in mouth. Salivation increased.
Head: Meningitic irritation: Subacute or chronic convulsions, epileptiform crisis, meningism during the attacks of fever (Proteus). For the attack on the kidneys and meningitis syndromes Sycotic Co C.M. and Proteus C.M. (Chebath Daniel)
Throat External: Goitre. Glands Posterior triangle enlarged.
Throat Internal: Hypertrophy tonsils and adenoids, and enlargement of glands of neck, chest or abdomen. Tonsillitis (recurring), cheesy masses from tonsil. Quinsy. Throat feels raw; scorched; dry. Profuse mucus from throat in morning. Swallowing difficult; chokes easily. Tracheitis.
Respiration: Asthma and bronchitis generally < with damp and frost and > at the sea side. Wheeze and cough_2_3 A.M. Wheeze and cough on waking. Hard spasmodic cough at night 2 A.M., 4 A.M., or 6 A.M. Irritable cough of croupy nature at night. The Sycotic child wakens at 2 A.M. with a cough of croupy nature. Attacks are spasmodic, affected by change in atmosphere, and may be prolonged. Cough till sick. Morning cough with easy sputum. Frequent bronchial colds. Bronchitis in winter. The respiratory mucous membrane is congested.with chronic 'juicy' or spasmodic persisting cough. Cough bad with tough sputum.
Neck and Back: Fibrositis_neck; shoulders and back. Backache severe; fibrositis back, myositis back. Stiff all over. Neuritis of head and neck. Sebaceous cyst back. Lumbosacral pain + +. Pain in loins. Pain sacroiliac joints. Hips stiff. Pain generally < after sitting, at night on beginning to move and > on moving, heat.
Desires and aversions: Aversion to: egg, fat, milk, milk pudding, cream, salt, sugar, vegetables, tea, vinegar, cheese, meat, bread, potato and tomato.
Upset by: egg < nausea, vomiting, bilious attack and hay fever. Thought of egg in morning produces nausea. Upset by fat, onion and oranges:
Averse to breakfast. Nausea with smell of cooking. Fond of : butter, fat, cheese, sweets, milk and salt.
Stomach: Nausea. Anorexia. Burning pain in stomach.
Eructation (acid); bilious attacks. Pain and distension in epigastrium. Flatulence. Nocturnal vomiting must empty stomach. Acidosis attacks.
Abdomen: Distended colon. Abdomen distended. Pain R.I.F. Pain L.I.F.
Digestive System: Chronic irritation of whole alimentary tract; catarrhal conditions; acute or chronic gastro enteritis in the child. Nausea or sickness after eating eggs (cf. *Ferrum met*). The children have digestive difficulties with a history of looseness of the bowel.
Circulation: Anaemia and hydraemia, usually in the adult.
Upper limbs: Fibrositis: shoulders. Neuritis: arms. Rheumatic pain in shoulders and arms; elbows and wrists. Arthritis of wrists. Arthritis fingers: pain generally > dry day and hot water. Fingers deformed; nodules on fingers.
Arthritis metacarpo_phalangeal joints. Arthritis especially of middle finger. Nodule between metacarpal 2 and 3. Fingers go dead; numb; with spasm of the fingers. Prickly feeling in hands. Nails brittle.
Lower limbs: Rheumatism of knees. ankles swollen and stiff + +. Soles of feet painful + +. Feet swollen at night. Big toe joint painful. Fidgety feet at night in bed. Feet and legs painful when walking, as if walking on loose cobble stones.
Neuromuscular System: General rheumatic fibrositis, aggravated from dampness, after a period of rest (cf Rhus Tox). Pain in the metatarsal bones. Fidgety feet.

SYCOTIC CO. continued

Urine: Irritation of mucous membranes from kidney to urethra. Albuminuria (high percentage of cases). urine: heavy smell. Nephritis; pyelitis; nephrosis; cystitis. Frequency and urgency with micturition. Urine corrosive. 'Urinary tantrums.' Kidney pain.
Genitalia: Menorrhagia and metrorrhagia. Polypi uterus. Amenorrhoea. Dysmenorrhoea. Leucorrhoea profuse and bland. Leucorrhoea: yellowish, white, dark brown, offensive and corrosive. The vaginal discharge is offensive and fishy. Pruritis vulvae; vulvo-vaginitis; balanitis; ovarian cyst; T.B. ovary and gland. mastectomy_malignancy. Impotency. Pain in left ovary at menstrual period. Genital warts and herpes.
Bowels: Constipation or looseness. Urgent call to stool as soon as rising out of bed. Loose, offensive stool, excoriating (cf. medorrhinum). Liquid motion after every meal. Motion pale, crumbly, bad odour.
Anus: Distended feeling in rectum. Splinter pain. Rectal prolapse. Perianal warts.
Sleep: Restless sleep. Night terrors. Nightmares. Grinds teeth in sleep. Perspiration + + head and body during sleep chiefly from 12 to 4 P.M. Insomnia. Wakes 2_3 A.M. Wheeze or cough. Can't sleep till 3 A.M. Dreams of dead bodies.
Skin: Sallow and oily skin. The typical skin eruption of Sycotic co. is warty: large, flat and rugged. The other typical Sycotic eruption is impetigo (especially children) Cracks on finger tips. Cracks on heels; taenia heels.
Circinate eruption on each arms. Nails brittle. Palmer dermatitis vesicles itch +, night <, heat <, excitement <, flour <, detergent < Wrists dermatitis cracks. Eczema on back of hands pustules with heat and itch. Circinate eruption of thighs and shins. Varicose eczema of ankles. Toe nails painful and brittle. Chilblains on feet < with heat. Paronychia. Herpes on face; neck and chest.
Herpes groin. Varicellar eruption of limbs since immunization. Intertrigo breasts. Eczema of face from 4 months to 2 years of age.

Pleomorphism

Paterson considered the sycoccus to be a pleomorphic organism. Many bacteriologists of the early 20th century believed that certain microorganisms could change their morphology under certain circumstances. It is a debate that has never been completely resolved, although pleomorphism was rejected by the majority of bacteriologists by the 1950s.

Over the last 30 years reports of extreme pleomorphism have continued to appear in the literature. Here is the conclusion of a paper by Milton Wainwright which has reviewed a number of these claims

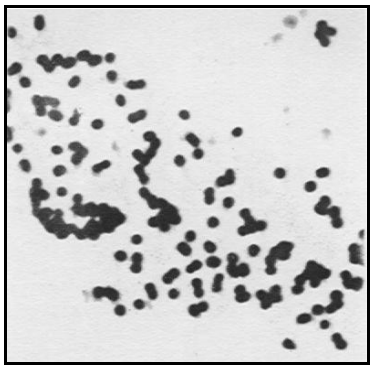
Conclusion

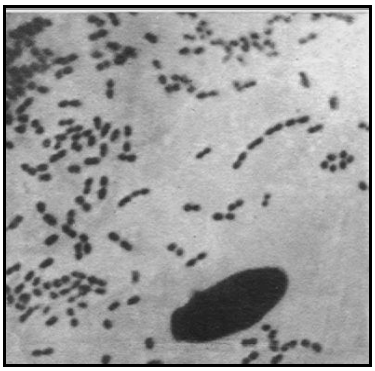
What can we make of all this? Most modern microbiologists, being monomorphists, would doubtless assume the examples of bacterial life cycles and extreme pleomorphism given here are merely the result of a mixture of wild speculation and contaminated cultures. (this was the view taken by Frobisher, who coined the word oligomorphism to describe the more readily acceptable examples, which clearly exist, of limited pleomorphism [20].) Yet most of the microbiologists who have reported examples of extreme pleomorphism went to considerable lengths to demonstrate the purity of their cultures.

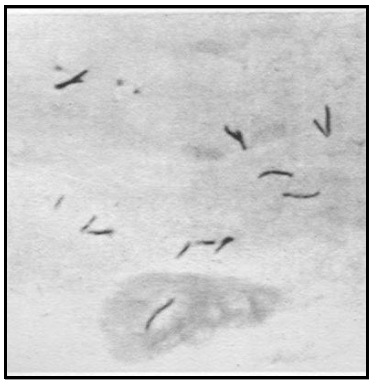
It is also worth remembering that they often spent more time-much more than most modern microbiologists do-just looking at bacteria. Likewise, they were generally more practised in the art of microscopy than are their modern counterparts. On the other hand, Holman and Carson showed that the work described by at least one researcher, who claimed to have demonstrated extreme bacterial pleomorphism, resulted from faulty bacteriological technique [32];.

Microbiologists of the past had no preconceived ideas about the nature of bacteria, and all possibilities were open to investigation. Of course, they lacked out technological sophistication - in particular, they knew nothing of the molecular approaches, which might be profitably used to study some of their apparently wild claims.

The literature on extreme pleomorphism remains intriguing, and some aspects of it may be worthy of reappraisal. By merely dismissing it, we may be ignoring something of fundamental importance. This is especially likely since examples of extreme variation in bacterial morphology continue to be linked with various diseases and cancer in animals and humans [33]. The use of molecular techniques should, however, help clarify any lingering uncertainties arising from the historical literature on extreme pleomorphism, although those certain of the phenomenon's validity would doubtless argue that their claims could be confirmed by simple, if thorough, microscopy. Perhaps it is now time to re-examine such claims with a non jaundiced eye.







Paterson’s photomicrographs of the Sycoccus, demonstrating pleomorphism.
The organism is seen morphing from a coccus to a bacillary form..

Wainwright , M. *Extreme Pleomorphism and the Bacterial Life Cycle: A Forgotten Controversy*---Perspectives in Biology and Medicine 40,407-414, 1997

Therapeutic Guidelines

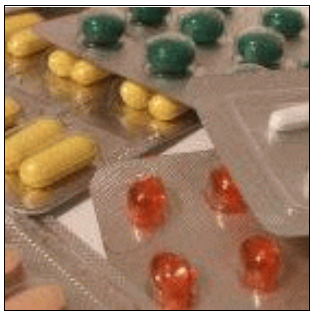
The dysbiotic case is a blocked case. Patient's whose intestinal ecosystem is significantly disordered have an on-board source of immunological and physiological chaos. If the symbiotic homeostasis is not corrected, the patient will be incapable of responding to a classical similimum. Or the response will be weak and short-lived.

One of the most important considerations for the physician is whether there are clinical features of dysbiosis. For the very experienced medical homeopath it may run against the grain to reduce the dynamics of a complex case down to a diagnostic label. If the diagnosis of intestinal dysbiosis is missed, however, the reactive features will not be enough to identify a cure for the case.

There are a number of features in the case history to look out for.

Key indications for the bowel nosodes

1. Aetiology: **infection, antibiotics or both**
2. Never well since... (**Acquired intrinsic blocks to cure**)
3. **Physiological / metabolic / immune corollaries** (signs of fatigue, debility, toxicity and vulnerabilty to infection). Prominent '**generals**'.
4. Self-perpetuating illness state (see dysbiosis - **systemic** cycle below) **Systems-distubances.**
5. Evidence of **altered surface immunity** (inflammatory conditions skin, mucus membranes, or internal integuments eg. synovium)
6. Symptoms referable to GI, GU, respiratory **tracts and body orifices** (although there are often persistent bowel symptoms, these can be surprisingly minor in comparison with the systemic corollaries)
7. **Insidious block to cure** (cases which are **failing to respond** to well chosen remedies, or where the patient consistently fails to build on an early response)
8. **Bacteriological evidence** of reduced lactose fermenting anaerobes, or evidence on stool culture of significantly increased populations of delayed/non lactose fermentors or pathogenic *enterobacteraceae*.



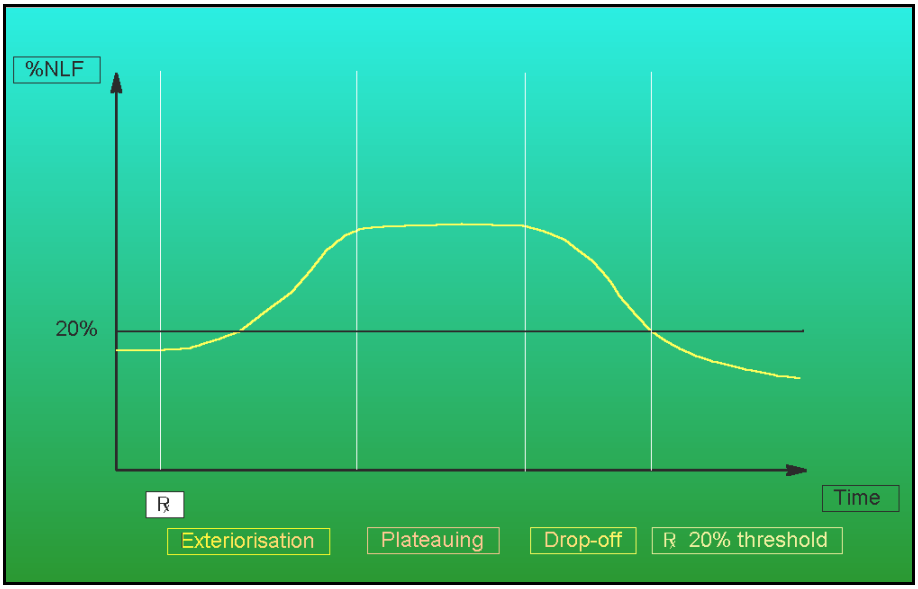
Drugs can give rise to dysbiosis quickly, or very insidiously.

Paterson's Clinical Guidance

"*...with regard to the change in the bowel flora [after a remedy]. The appearance of non-lactose fermenting organisms, I regard as evidence of the action of the defensive body mechanism. Their percentage in relation to B. coli and their persistency in point of time may be used as an indication upon which to base treatment at any period of the disease*".

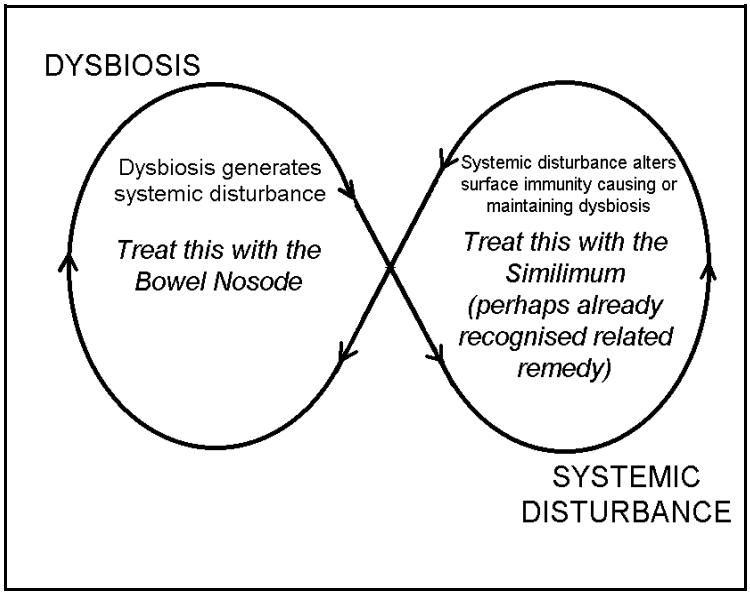
"*If the percentage is high (80-100%) clinical experience has shown that the potentised vaccine (nosode) does definite harm*". [May 'block' an acting remedy.]

"*Now with a positive stool yielding 20% or less, I should not hesitate to use the corresponding nosode or autogenous vaccine, provided the patient does not show other evidence of improvement*".



After a remedy there is an increased presence of non-lactose fermenters in the stool.

Therapeutic Guidelines continued

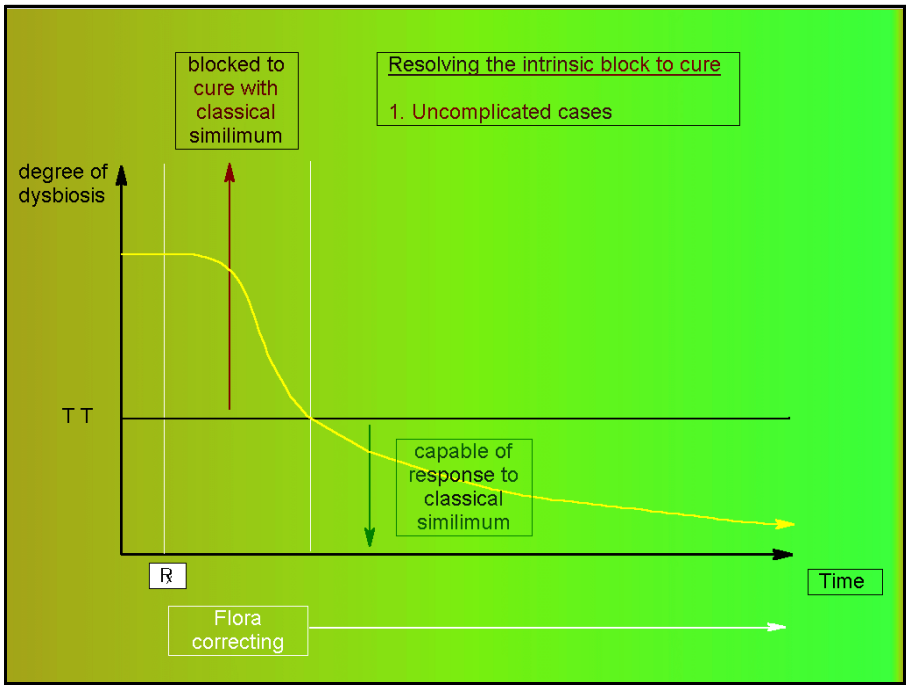


The general consensus in the literature is that the Bowel Nosodes do not stand repetition. They are given as stat doses, or split stat doses, over one or two days. The author prefers three stat doses, in rising potencies, over twelve hours.

The traditional advice is then to wait, and to avoid repetition of the nosode within 3 months.
In my experience the patient usually some shows some evidence of a response within 10-14 days after a good prescription. (Sometimes earlier)

Where the bowel nosode is used on its own account, as the main therapeutic input, I would leave the resolution to unfold in an open-ended way (weeks), if they are showing ongoing improvement.

In uncomplicated cases the patient's intrinsic block to cure will resolve and they will become responsive to a classical remedy.
The indicated similimum should be given if they plateau in their clinical response.
The diagram opposite is a representation of resolving dysbiosis after a bowel nosode, showing the threshold beyond which a remedy response can occur.

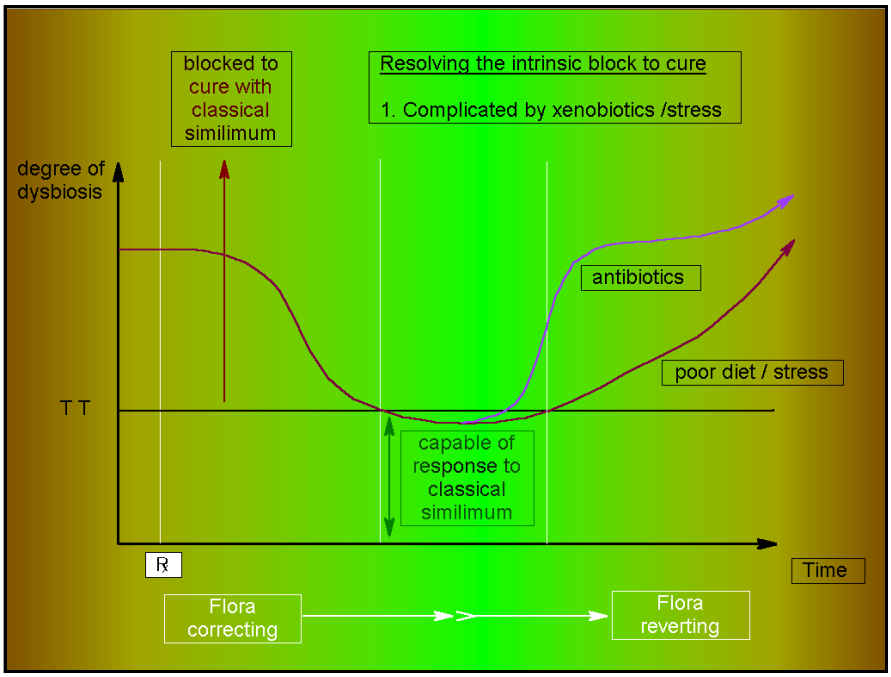


When the bowel nosode is being used to resolve a block to cure, or augment the response to a partially effective remedy, I would leave 14 days or more between the nosode and the related remedy.

My rationale for this is that many chronic cases show two or more main cycles of causation, and these may need to be resolved sequentially to achieve progress.

Therapeutic Guidelines continued

In some cases if you wait too long after the nosode to introduce the similimum, the systemic disturbances will re-evoke the intestinal dysbiosis. If you do not wait long enough between nosode and similimum the dysbiosis will continue to block the remedy response. In children the time lapse is shorter than in adults. Adults with longstanding active bowel symptoms and debility should be left longer to respond.



A well chosen bowel nosode does not appear to be blocked in its response by a well chosen similimum. However, a well chosen similimum which is slowly resolving a long-standing illness, may be blocked by early repetition of itself, or of its related nosode.

In cases which you have successfully 'unblocked' and which are resolving with the similimum, it is best to follow the traditional advice and avoid repetition of the nosode, unless there are clear indications that the bowel symptoms are re-emerging and the patient is deteriorating clinically.

NB Discharges, catarrhs and eruptions in the post-similimum phase of treatment are not indicators of worsening surface immunity. These features are all too frequently treated by orthodox prescribers with antibiotics - often rendering the patient dysbiotic once again and returning them to their state of fatigue or debility.

In infective acutes the early use of the correct similimum will prevent dysbiosis emerging sub-acutely. In sub-acute infective cases, the indicated nosode can be used alternately within a series of similia, in high potency, which reflect the dynamic changes in the current state of the patient.

Many patients showing signs of dysbiosis have had two sequential courses of antibiotics within a short time frame. (usually with different spectra of antibactial activity). If they have also had treatment with antipyretics they may show signs of thermostatic instability and fatigue. In this event use a physiological similimum at an early stage of treatment. (See rubrics for fever suppressed / remittent; or rubrics relating to the abuse of quinine.)

There is plenty of room for error in the selection process for a bowel nosode on purely clinical features. Even careful symptom-analysis using a bowel nosode repertory like the one given in this book can lead to the wrong choice of nosode. If the patient fails to respond, but conforms to any the criteria on page 5.1, it would be wise to try another bowel nosode before moving into an entirely new field of prescribing.

Clinical Remedy Relationships

Notes on the Bowel Nosode Relationships

The materia medica of the bowel nosodes has been worked out in the clinic over the course of several working lives. A major element in the treatment of chronic cases is in the process:

- clinical exploration
- development of models for the illness
- engagement with the available treatment data
- selection and timing of treatment
- re-evaluation and adjustment of models, analyses and treatment
- feedback into the fund of clinical data and teaching of others

These cycles of clinical feedback generate information of potential value to other prescribers. In studying and using the bowel nosodes, there are several ways in which historical clinical information can be helpful:

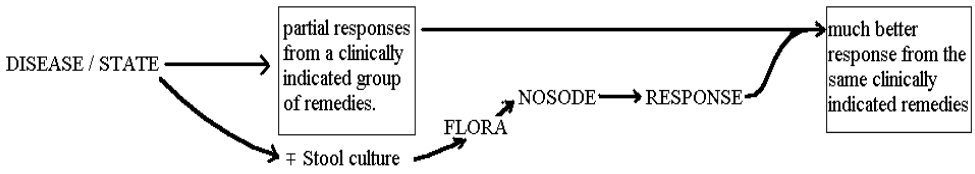
- providing additional information governing choice of nosode
- understanding the relationship of the nosode to other treatments
- informing their timing and placement within the treatment programme

In the early days, the bacterial composition of the stool was an important factor in guiding treatment choices. Stool composition changes under the influences of:

- illness
- diet
- drug treatment
- the clinical homeopathic similimum
- the indicated nosode

In the ill patient with bowel dysbiosis, use of the clinical similimum or constitutionally based remedy appears to evoke host responses and a shift in the surface immunity in the bowel.
In the clinical experiments of Bach and Paterson, the number of non-lactose fermenting organisms was frequently observed to increase in the stool, for a time after homeopathic treatment (perhaps as bacterial surface adherency diminished). This shift in flora was associated with clinical improvement.

The observed shift in the bacterial composition of the stool appeared to bear some relationship to the remedy used. As time went on, this quasi-objective information was collated and became the first major influence on remedy-nosode relationships.



It is clear that much of the existing data suffers from some obvious limitations in mid twentieth century microbiological knowledge, and all these basic hypotheses really need to be reinvestigated. It is also clear that many other variables may be operating in these complex clinical situations, and control group comparisons are not available, so we have to be careful not to rely entirely on these observed associations.

As the body of knowledge and experience increased, clinical outcomes became the main method of establishing remedy-nosode relationships. 'Blocked' cases or cases which had plateaued in their response would be found to improve after the use of an appropriate nosode. Their response to the similimum or constitutionally based treatment would then improve, and the empirical relationship between nosode and remedy would be documented. Clinicians like Wheeler, Dishington, Griggs and Elizabeth Paterson [38] have been very influential in reporting cases and gradually extending these clinical relationships. (see table 18). I have found this information very useful in the clinic and there appears to be more than a little truth in these observed relationships, although proving them statistically is an entirely different matter!

Looking at the remedy list (in table 18) it is obvious that there are hundreds of remedies in general use among experienced homeopaths, for which a nosode relationship is not established. Today we have access to remedy data that was much more difficult to access in years past. So it is possible to synthetically repertorise on the key clinical information available for each nosode and explore possible relationships further. It is also interesting to see whether 'known' relationships are bourne out by the repertory.

On the pages that follow there are a series of experimental repertorisations. Symptom information from *'A survey of the bowel nosodes'* by Elizabeth Paterson [38] has been entered in various combinations and the rubrics analysed.

Symptom groupings have been analysed on various rationale:

- 'totality' (selection of the most consistent contextual information)
- 'essence' (key mind rubric and consistent contextual and local information)
- 'pathological' (key rubrics for surface-immunity, system or locality)

The resulting analysis for each nosode usually contains 'established' clinical relationships and also lists a variety of possible relations that have not yet been confirmed clinically. Some nosodes (most notably Proteus) do not align very convincingly with established relationships and a short impression for each analysis is given on the pages that follow.** This section is now available on CD.**

The rest of this section is made up of a series of tables which bring together 'established' and 'notional' relationships. I have annotated the entries to show those that have been 'confirmed' in my experience, together with some theoretical relationships bourne out of the repertory search. A few of which are annotated to show which of these 'unknown' relationships appear to have worked for my patients. **This section is now available on CD.**

Bowel Nosodes and the Mind

There is little doubt that homeopathy has tended to place the mind at the centre of the case since the time of Kent. The prescribing data should be placed in the context of the prevailing Kentian methodologies of those who did most of the seminal work on them. This raises several questions on the nature of the mind symptoms attributed to the bowel nosodes:

Do the mind features represent attributes that drive the case towards a particular kind of dysbiosis?

Does overgrowth of a particular organism accentuate certain mental/ emotional symptoms?

Are there any psycho-immunological models that help to explain mind phenomena associated with these nosodes?

Have the mind symptoms in the literature been projected onto the bowel nosodes from those of their apparently related remedies?

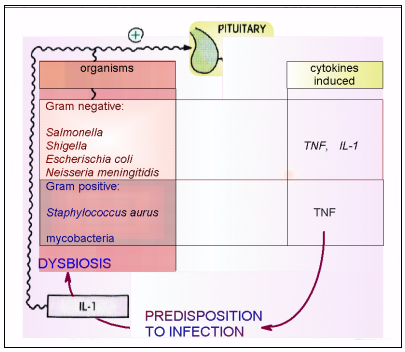


Fig 47

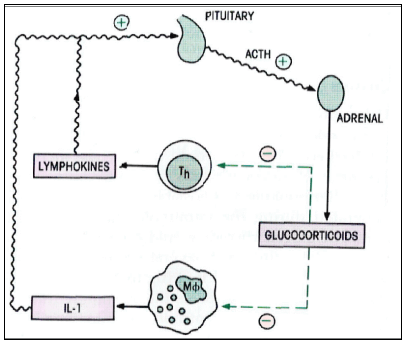


Fig 48

Most of these questions are difficult to answer in a concrete way. We will briefly examine some possible immunological models for some of the central effects that occur in infective and dysbiotic states. It is thought that a number of cytokines have neuro-endocrine effects which may alter mood and the pituitary adrenal function.

Some gram negative organisms (including several implicated in dysbiosis) release lipopolysaccharides which induce TNF, interleukin-1. (Fig. 47)

The presence of these compounds is associated with bacteriologically mediated inflammatory responses and if their levels are chronically raised, as a result of dysbiosis, they may reduce immunological efficiency and predispose to secondary infection. (Fig. 48)

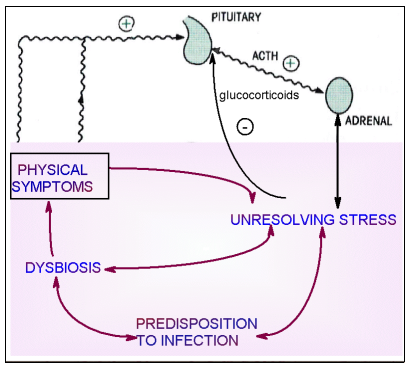
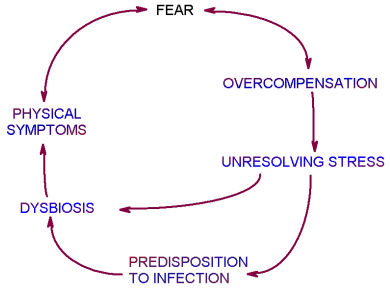


Fig 49

IL-1 stimulates pituitary function and evokes a biochemical stress response. In the acute infection a short term positive increase in adrenal function is probably immuno-stimulatory. However, protracted increases in adrenal activity ultimately inhibits cellular immunity. We observe the same phenomenon in people who are chronically stressed. (Fig. 49)

The question of whether emotional disturbance predisposes to dysbiosis appears to tenable in terms of mind-body relationships and the foregoing observations. Whether certain mental/emotional themes predispose to specific kinds of dysbiosis is a tantalising but purely speculative idea at the present time. Here are some thoughts:



Bacillus 7 - driven by material ambition or fear of insolvency - work
Bacillus 10 - driven by fear of aging or losing sexual allure - sex
Dysenteria co. - driven by anxiety of conscience - self worth
Gaertner - driven by awareness of frailty and the need to make a mark - creativity
Proteus - driven by unremitting environmental stress - chronic autonomic overdrive
Sycotic co. - driven by shame (try to compensate for their dirtiness) - infected
Morganiae - driven by greed for the good life
Faecalis - driven by the desire for understanding

Bowel Nosodes and the Repertory

The data

At first glance, the remedy data for the bowel nosodes seems too vague and general to be of clinical value. The leading symptoms and keynotes can rarely be classed as 'strange', or 'peculiar'. So bowel nosodes are not usually 'jumped to' on the basis of a single strong feature in the case. A variety of inductive methods (based on the context) are required. It may also be necessary to undertake some form of analysis using the available clinical data.

Empiricism

The data for the remedies themselves is highly empirical. Most of the patients, for whom they have been prescribed in the past, have been chronically unwell, or at least sub-acute. The remedies themselves cannot be said to have undergone a standard proving, although clinical observation and stool culture data lends some objectivity to Paterson's case series.

The priorities of Bach and Paterson were, as far as possible, to establish a scientific basis for the selection of bowel nosodes. Whether it was this priority, or a general lack of keynotes, (in what was a chronic and often debilitated patient sub-population), we find that 'leading symptoms' for the bowel nosodes are in short supply. However, what information there is, is in my opinion, more reliable than much of the proving data in the materia medica as a whole.

Repertories

Modern repertories have imported the bowel-nosodes into their rubrics, but no one appears to have marked up the rubric entries as they are clinically verified, in spite of the considerable number of cases that have appeared in the journal literature over the last fifty years. As a result, the nosodes have never been elevated above 'normal type' in the standard repertories of the day.

This low-key representation, together with the small overall number of symptoms, means that these nosodes never turn up in a totality analysis. 'Broad sweep' repertorisations, which analyse only large headline rubrics, do not bring them out. Expert systems and family group searches fail to show them up, even in those patient analyses where they are clearly indicated and ultimately shown to be effective.

Given this poor representation, it is wise to do two repertorisations in those cases where the bowel nosodes are clearly indicated.

- in one repertorisation you would use traditional methodology (whether it be totality, thematic, pathological or synthetic) and establish the range of potential similia.

- in the other repertorisation you would use a nosodes repertory to assess which remedy is most likely to address the *systems disturbances* relating to the patient's dysbiosis

As you become more familiar with the nosodes, these two repertorisations will inform one another. So, for example, if your 'traditional' analysis yields *Phosphorus*, *Silica* or their salts, you will probably use the nosodes analysis to assess whether *Gaertner* is indicated.

With experience you will come to use these empirical relationships to good effect, using the remedies sequentially to 'unblock' the case or augment the response of each to the other.

We have included a recompiled bowel nosode repertory (on page #), which uses the search-word and chapter conventions of modern clinical repertories. Because the listings are short, it takes only a few minutes to do a hand repertorisation on the nosodes.

Analysis methodology

Unless you are very clear that an uncommon symptom is unique to a remedy you would be wise to keep the analysis general and favour the head rubrics. The more unusual the feature, the more likely that the data is derived from a single case study, and potentially the same symptom could arise from time to time in patients who are sensitive to a different nosode.
So beware, don't use small rubrics to exclude remedies. Use them only to lend support. Nosodes which do not appear in the listing for a common feature are easier to exclude.

Beware that 'small' nosodes like Bacillus-10 are severely under-represented, even in a highly selective bowel nosode repertory like this. It has been used very rarely and has therefore generated much less data than its counterparts. If an analysis throws up three points of contact with Bacillus-10, as opposed to six for Morgan pure, you should consider Bacillus-10 quite carefully and read the materia medica of the remedy.

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