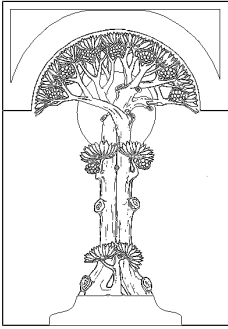


Centre for Integrative Medical Training
In Association with London Integrated Medical Health Education



Pre-membership Course in Medical Homeopathy

A Blended Course in Homeopathic Medicine for Healthcare Professionals

Unit 66

PRINCIPLES & PRACTICE - methodological discussion notes for Week 10

NOTES on Environmental Aetiologies in Chronic Illness

Factors affecting the relative balance of local versus systemic effects of a substance.

The route of entry of a substance will often show maximal damage if the substance has a high intrinsic activity (see earlier sections on the acids).

It is for this reason that much of the homeopathic materia medica has associations with the skin and upper GI tract. This skewing of the data comes from the fact that sections of our remedy data comes from accidental or intentional 'poisonings' (including proving experiments*).

Materia Medica - Data Sources:

- A. High potency provings
- B₁. Toxicology
- B₂. Low potency provings - material doses (some could be considered toxicological)
- C. Clinical experience

High potency provings (Group A above) yield energetic/physiological/mind remedy data.

We have already discussed the 'constitutional' picture of Fl-ac, for example (Sheila Y case study). In this case our 'high potency' remedy indications showed only a superficial resemblance to the toxicological description of the remedy in the homeopathic materia medica.

Poisonings (B₁) - and, to a lesser extent, **low-potency provings** (B₂)- yield:

1. **Local toxic symptoms** often on the contact tissue, or at the route of administration.
(eg. Fluoric acid causes severe local tissue breakdown at points of exposure)
2. **Systemic symptoms**: caused by disturbed physiology at a cellular or cytoplasmic level.
(eg. Cadmium blocks oxidative phosphorylation stopping ATP formation)
3. **Sub-systemic effects** of toxic disruption of neural/endocrine control pathways.
(eg. Organophosphates cause cholinesterase inhibition and peripheral neuropathy)
4. **Sub-systemic effects of organ toxicity**: eg heart, kidneys, Peripheral nervous system
(eg. Tetrodotoxin is a neurotoxin from the puffer fish. It attacks peripheral nerves at the node of Ranvier, causing sensory paraesthesia of the mouth and limbs followed by paralysis.)
5. **Local manifestations** of 3 & 4 above.
6. Sequelae of immunotoxic effects
7. Carcinogenesis

In disease, most of the symptomatology arises from the host's attempts to compensate, so we should try to guard against using toxic remedy symptoms from Group 1 above, but should try to match local effects of the wider host response (2-5) rather than local toxic effects.

The exception is *homotoxicology* where all the host's local symptoms are interpreted as toxic effects and treated with an isopathic preparation of the toxin, to promote its elimination.

* Clinical pathogenic trials

Homotoxicity

The toxicology of daily living is grossly overlooked by doctors, industrialists and politicians.

“The greatest improvements in the productive powers of labour... seem to have been the effects of the division of labour” Adam Smith *Wealth of Nations* 1776

Specialisation and sub-specialisation have indeed multiplied the processes of manufacturing and supply, to the extent that no-one has any power of veto in the modern “laissez faire” economies. Legislation and control is only switched on once the toxic consequences of a commercial activity are underway and the evidence has been presented - usually by poorly resourced pressure groups.



<https://www.sciencedirect.com/topics/medicine-and-dentistry/environmental-toxin>

Important human neurotoxicity outbreaks

Toxin	Year	Location	Magnitude (reported clinical cases)
Tri-ortho-cresyl phosphate	1959	Morocco	> 40,000
Mercury	1971	Iraq	6,000
<i>n</i> -Hexane	1963	Japan	93
Methyl <i>n</i> -butyl ketone	1973	USA	68
Clioquinol	1963	Japan	several thousand
Kepone	1975	USA	76
Dimethylaminopropionitrile	1978	USA	104

from *Neurotoxicology*: John L. O Donoghue

Estimated numbers of contact agents

1,500 active ingredients of pesticides ^H

4,500 active ingredients of drugs ** ^H

2,000 drug additives to improve stability, inhibit bacterial growth etc. ** ^H

2,500 food additives with nutritional value *

3,000 food additives to promote product life * ^H

50,000 additional chemicals in common use (*) ^H

from Hodgeson E; Guthrie F.E.: *Introduction to biochemical toxicity* (elsevier/North Holland, NY 1980)

** Categories in which doctors have direct influence

* Categories in which doctors have an advisory or research role

(*) Categories in which doctors may have limited case by case involvement

^H Categories in which homoeopathy may have a role (isopathy, tautopathy, homoeotoxic)

We will return to the subject of isopathy/tautopathy/homotoxicity in the sections on immunity and cancer.

MODELLING in the Homeopathic Treatment of Co-morbidities in Chronic Care

As physicians we have three kinds of input in respect of illness:

1. Prevention
2. Treatment
3. Rehabilitation (which may involve ongoing input of 1 & 2)

1. Prevention

Prevention is obviously the prime objective and requires:

- awareness of intrinsic and extrinsic risk factors
- awareness of trigger events to decompensated states
- awareness of treatments which facilitate full host recovery from acutes preventing chronic sequelae
- awareness of treatments which can undermine host responses and increase the risk of chronic states

2. Treatment

Treatment of acutes can be:

1. Conservative: reassure the patient and wait for spontaneous resolution in a self limiting condition.
2. Homoeopathic: a homologous stimulus to facilitate self-correction.
3. Allopathic: targeted at causation (eg. antibacterial) while host compensation/defence occurs. (!)
4. Enantiopathic: local or general symptom modification (!!)
5. Enantiopathic: control (or 'suppression') of local or general reactions (!!!)
6. Compensatory: systemic support in critical / life-threatening acutes

Treatment measures 1 & 6 above are rarely controversial when appropriately applied on the basis of an accurate diagnosis and prognostic assessment.

Serious differences of opinion surround the use of measures 2-5 above.

3. Rehabilitation

This is more controversial than we are often aware, as exemplified by the differences between the orthopaedic and osteopathic approach to back injury. Or the marked differences in management of frozen shoulder between acupuncturists and orthodox general practitioners. Stroke rehabilitation has only recently begun to incorporate methods of maximising neurological 'plasticity' / adaptability.

Our first duty is always, however, to do no harm. Unfortunately, as mentioned on page #, we have around 6,500 drugs, potentially, with which to do unintentional harm...

Identifying potential Iatrogenic factors in the Chronic Case

Understanding the potential for iatrogenic illness has implications for prevention, treatment and rehabilitation. The following case scenario will be discussed:

Mr J N is 57 years old and used to work in a factory in the central belt of Scotland. His workplace exposed him to solvents, paint-fumes and airborne hydrocarbons. He developed wheeze, and was placed on bronchodilators. He fell out with the safety officer at work and became very indignant about his working environment. His wheeze became suddenly much worse. He was placed on steroid inhalers and began to get chest infections. His respiratory function deteriorated and he began to lose time off work. He was started on oral steroids and later contracted aspergillosis in a damaged area of his right lung. The steroids caused weight gain and reduced his exercise tolerance. He developed musculoskeletal pain and started to use paracetamol regularly. The chest infections became more frequent. He developed night sweats and intermittent low grade fevers.

He was paid off and entered an unsuccessful action for compensation in respect of occupational illness. He became embittered and angry and his chest deteriorated further. He was issued with a home nebuliser. He developed a steroid induced ulcer and was commenced on H2 blockers. He became depressed and was commenced on antidepressants. He suffered a collapse of a midthoracic vertebrae due partly to his increasing weight and partly to steroid induced osteoporosis. This caused marked pain on coughing and increasing reactive spasm in the intercostal muscles. He was commenced on diazepam 2mg prn to allay the spasm and attendant anxiety. Mr JN was then referred (reluctantly) to the networked NHS homoeopathic services.

Treatment 1. Homoeopathic Cortesol 200c stat. - no change

Treatment 1b. Sulphur 30, 30, 200 stat/3 5 days after Cortesol - short term reduction in wheeze

Treatment 2. Aspergilla 30, 30, 30 - caused aggravation in wheeze requiring immediate increase in orthodox medication (cf. Clinical case IS parts 2, 9)

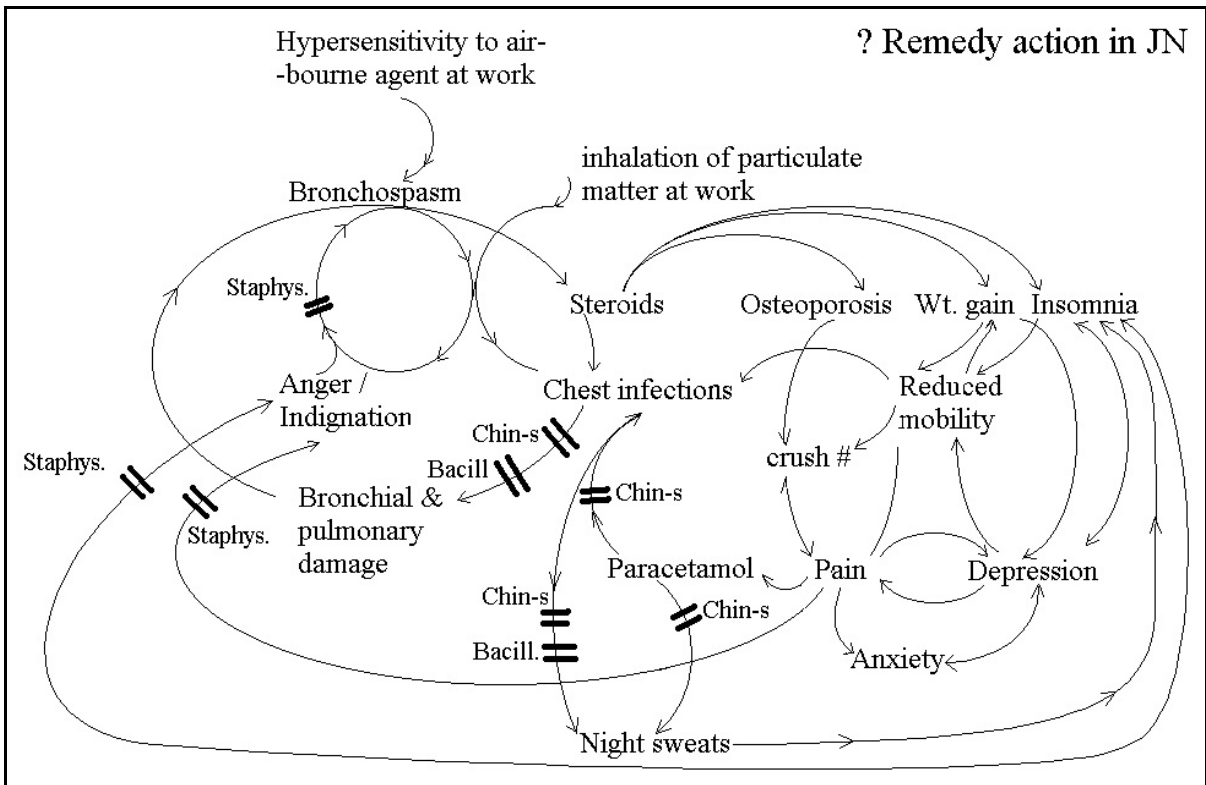
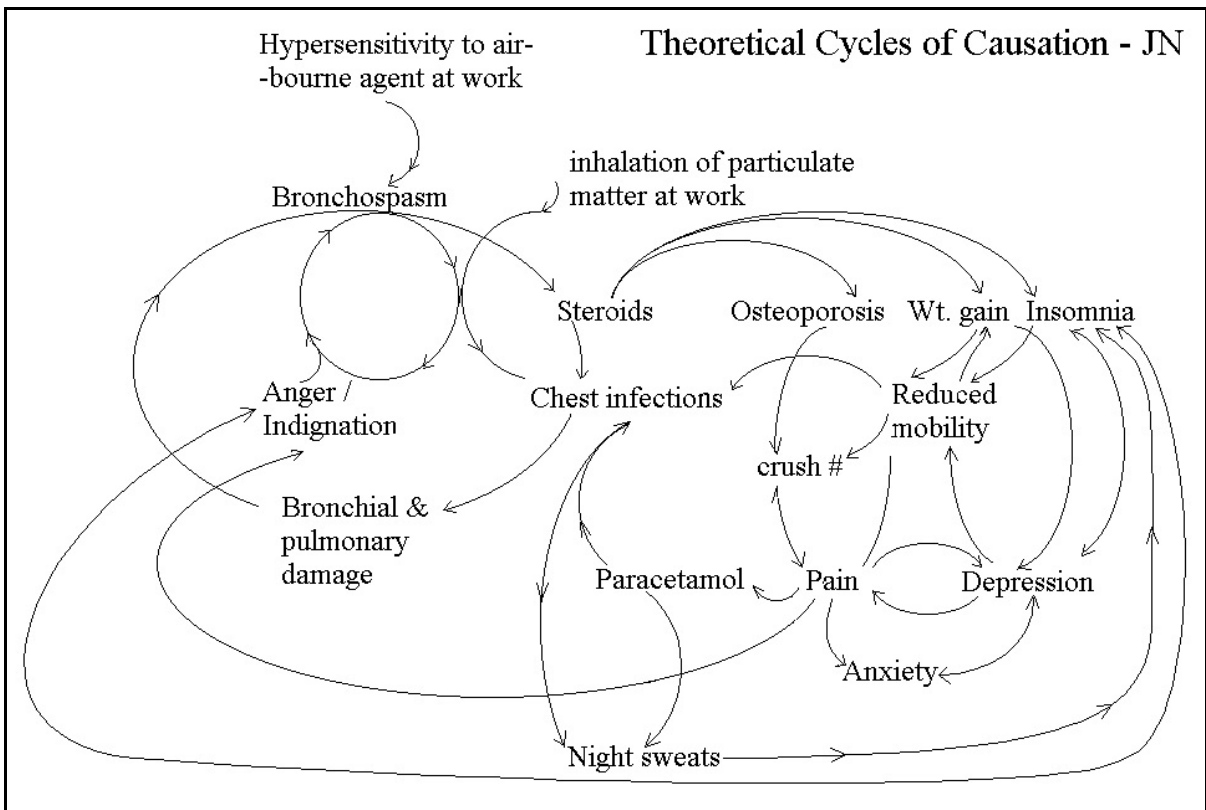
Treatment 3. Nebulised China-s 30c - marked improvement, wheeze settled and expectorated casts and plugs of chronically retained secretions. Incidence of chest infections settled. No antibiotics for weeks, reducing steroids. Developed an acute bronchitis - phoned. Reducing paracetamol on my insistence.

Treatment 4. Bacillinum 30c/6: rapid improvement, no antibiotics required for the acute (for the first time ever). Still troubled with chest pain. Still also wheezy and tight in the chest on minimal exertion. Expressing indignation about the way he has been treated by his firm (and everyone else). Nevertheless reducing steroids gradually.

Treatment 5. Staphysagria 30/200/1M: tremendous improvement over several weeks. Spasms in chest >, wheeze >, much less internalised anger and indignation. Gradually increasing exercise. Stomach symptoms reducing. Weaning himself off antidepressants. Steroids still reducing.

Treatment 6. Mother died. Going through a bereavement. Chest mildly worse but hasn't required to increase any of his orthodox medication. Issued with Nat-s. 30,30,200 but told not to take it unless his chest deteriorated further. To continue steroid reduction.

March 1998: Discontinued steroids, paracetamol, antidepressants, nebuliser. Using ventolin prn. To try off H2 blockers and diazepam. Beginning to lose weight gradually.



Discussion

In the case of JN the orthodox emphasis was on “management” and “control”. Additionally his own inclination was to control and internalise his anger. You may not agree with the cycles of causation that I have proposed for his condition. Nevertheless, you might agree that his orthodox drug treatment has been of considerable influence in the final expression.

Firm proponents of enantiopathy and allopathy would argue that Mr JN might have died of bronchopneumonia long since, had he not received the drugs he did. Homoeopaths might argue that proper safety equipment at work and appropriate acute prescribing would have prevented the evolution of the sub-acute state altogether.

Prevention is better than cure

Let us return to the **prevention** of self-perpetuating systemic illness. You will recall that we identified the following:

1. awareness of intrinsic and extrinsic risk factors
2. awareness of trigger events to decompensated states
3. awareness of treatments which facilitate full host recovery in acutes, preventing chronic sequelae
4. awareness of treatments which can undermine host responses & increase the risk of chronic states

1. Intrinsic risk factors

These can be genetic or hereditary - as elucidated by modern epidemiology and genetics. Or they can be miasmatic. For example in HIV, the underlying vulnerabilities may determine how AIDS manifests:

Miasm	Co-factors	Extrinsic	Pathology
Tubercular	Mycobacteria	Smoking	Pneumocystis carinii pneumonia Mycoplasma aviare
Carcinoma	Multiple	Carcinogens	Non-Hodgkins lymphoma / Lymphoma
+ Psoric	Herpes		Carcinoma skin Histioplasmosis Kaposi's sarcoma
Syphilitic	Toxoplasmosis CMV Sexually transmitted infections	Drug / alcohol abuse	Leukoencephalopathy / Cerebral atrophy
			Neuropathy
			HIV cardiomyopathy
			Retinal microaneurysms
Sycotic	Dysbiotic states Poor diet	Antibiotics Suppressive topicals	Hairy oral leukoplakia
			Molluscum contagiosum
			Pyogenic granuloma
+Psoric			Enteric Candidiasis
Psoric	Herpes Staph/Strep/Fungi	Steroids Immunosuppressants	Psoriasis Multidermatomal shingles



<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4238727/>

<https://bmcinfectdis.biomedcentral.com/articles/10.1186/s12879-020-05593-4>

2. Extrinsic risk factors:

These can range from environmental pollutants to stressful domestic environments. For example, if someone develops unheralded mental illness with uncharacteristically violent or chaotic behaviour, their partner - in trying to suppress a like response - is very likely to enter into a *Staphysagria* state.

If recognised, the homoeopath can prevent the emergence of neurodermatitis, recurrent UTI, musculoskeletal pain syndromes, hypertensive states, asthma etc.

3. Triggers to decompensated states:

You are already aware of these: Accidents, Bereavements, Shock/Fright, Infections, Toxic exposure, Surgery, Disappointments, Major life events, Invasions, Loss, Sexual abuse/Rape, etc...

4. Facilitation of recovery from acutes:

This is essential to the avoidance of sub-acute and perpetuating illness. The aetiology is important, and the remedy in most acute states will be a plant remedy. The acute picture usually reflects the dominant toxic alkaloid (A). You are already aware of the acute pictures for most of the following*.

Acute	Compare → Fine tuning → Subacute relations			Fundamental relations
Aconite *	Other aconites	Aconitine (A)	China-sulph (?)	Sulphur
Arnica *	Other compositae			Graphites (?)
Belladonna *	Other solanaceae	Duboisia	Atropinum (A), China	Calcaria carbonica
Chamomilla *	Anthemis, Puls.	Cypripedium	Convolvulus / Jalapa (?)	Magnesia carbonica
Camphora	China officinalis		Baja, Pambotano	Hydrocyanic acid
Colocynthis *	Curcubitaceae		Rheum (?)	Cuprum (?)
Digitalis	Nerium odorum	Adonis, Crat.	Ichthyotoxin	Kalium (?)
Gelsemium *		Baptisia		Mag- phos., Phos-ac. (?)
Helleborus *	Other hellibores	Polymnia	Iodoformum	Zincum
Hyosciamus	Other solanaceae	Hyoschine (A)	Scopolamine (A)	Alumina (?)
Ignatia *	Cimicifuga		Strychninum (A)	Natrum muriaticum
Lobelia *	Other lobelitaceae		Tart. emeticus	Arsenicum album (?)
Opium *			Codeinum (A)	Carbo-veg (?)
Phytolacca *		Arum, Bryonia		Mercurius
Pulsatilla *	Cham., Cycl.,	Anagyris,	Atriplex, Penthorum	Silica
Staphysagria*	Ign., Anan.	Caladium	Phos-acid	Causticum
Stramonium *	Solanaceae			Phosphorus
Tabaccum *	Veratrum album	Nicotinum (A)	Hydrobromic acid	Acids
Veratrum vir.	Gels., Bapt., Bell.,	Usnea	Glonoine	Ferrum phosphoricum

