Antropologia medica degli eventi dell'infiammazione acuta e cronica
"Il fuoco che ringiovanisce"
Roncegno,12 ottobre 2006

Relatore:dr.Giancarlo Cimino

Giordano Bruno.

Ab umbris ad ideas

1.Purgatio animi
2.Cognitio operum divinorum
3.Contemplatio ordinis
4.Comparatio.
5.Negatio.
6.Oratio.

Ut amore boni concitus ex statu intellectuali transformeris in bonum superius intellectu.

Premessa epistemologica

Goetheanismo	Atomismo
La realtà è data al soggetto umano come	
Sintesi di percezioni e concetti operata dall'attività pensante.	Analisi delle percezioni intese come rappresentazioni.
La realtà è fatta di "stati" che si presentano al soggetto in determinate condizioni di	La realtà è fatta di atomi tenuti insieme da moti di attrazione e

Premessa epistemologica

Goetheanismo

Atomismo

Mondo Inorganico

Fenomeni archetipici = Leggi naturali deterministiche

L’idea(archetipo) è esterna al fenomeno

Mondo Organico-vivente

Osservazione comparativa dei tipi

Interazione geni/ambiente

L’idea (archetipo) opera nel fenomeno

Uomo

Presenza di un vissuto biografico

Interazione geni/ambiente

L’idea (archetipo) si esprime come autocoscienza

Evoluzione culturale

Epistemologia del fenomeno infiammazione.

L’infiammazione è una realtà ricavata dalla sintesi di dati analitici (percezioni)
e concetti che via via compenetrano e organizzano questi dati.

I dati si ricavano sia dall’osservazione macroscopica (es.Rubor, Tumor etc.)
sia da quella microscopica strumentale (isto-citologia, analisi biochimiche e biofisiche)

“Il processo infiammatorio è un processo difensivo” (Virchow)

“Nel processo infiammatorio si distinguono:
1) Una fase di eccitazione delle terminazioni nervose periferiche;
2) Una fase di iperemia attiva;
3) Una fase di ischemia relativa;
4) Una fase di iperemia passiva.” (Ricker)

“Ad una fase catabolica che avviene immediatamente dopo l’azione dello stimolo infiammatorio,fanno seguito processi anabolici”.(Husemann-Wolff)

“All’interno di questa parola chiave, infiammazione,mettiamo realtà fra loro diversissime. Processi e meccanismi che hanno sì, qualcosa in comune,ma anche differenze profonde.
Ecco perché sta emergendo il concetto di “forme polarizzate di infiammazione”
Infatti i processi infiammatori sono estremamente diversificati, e a questa diversificazione corrisponde un diverso assetto dei programmi genetici delle cellule che orchestrano le reazioni infiammatorie. E’intuitivo che per il sistema immunitario ben diverso è affrontare un grosso parassita extracellulare…rispetto ad eliminare un germe intracellulare…Ebbene , a questa diversità di problemi l’organismo risponde con forme di infiammazione differenti, orchestrate da cellule fagocitiche , dette macrofagi.”

Insieme al concetto di fenomeno difensivo porre la questione:
Infiammazione e crescita

Nel processo infiammatorio acuto, la fase anabolica finale è affine ai processi di crescita.
(scatto accrescitivo dei bambini dopo un episodio febbrile)

Crescita di un cristallo	salificazione
Crescita vegetale	totipotenza direzionale ed organica.
Crescita animale	differenziazione, apoptosi ,

Infiammazione e processi del sangue

Nell’organismo umano osserviamo: un polo freddo, neurosensoriale
e un polo caldo, metabolico

Il Sangue e la respirazione garantiscono l’omeotermia e l’equilibrio fra i poli

Se compare una tendenza raffreddante –cristallizzante (polo freddo) nell’organismo animale ed umano si ha una risposta infiammatoria (polo metabolico) riequilibratrice.

Infiammazione: tendenza alla localizzazione o alla generalizzazione

Il sistema immunitario può essere attribuito in parte al polo della forma-freddo
(neurosensoriale) (fenomeni riparativi e limitanti l'infiammazione), in parte al polo caldo (metabolico) dell'organismo.

La tendenza alla generalizzazione dei fenomeni infiammatori è inversamente
proporzionale alla forza espressa dal sistema immunitario.

D'altro canto la risposta infiammatoria stessa può essere concepita come direttamente proporzionale allo stimolo raffreddante.

Essa tende a far "oscillare il pendolo" eccessivamente nella direzione del calore.

Il medico è il supervisore e l'accompagnatore del processo con la

Clinical Aspects of Acute Inflammation

Redness (rubor)

An acutely inflamed tissue appears red, for example skin affected by sunburn, cellulitis due to bacterial infection or acute conjunctivitis. This is due to dilatation of small blood vessels within the damaged area.

Heat (calor)

Increase in temperature is seen only in peripheral parts of the body, such as the skin. It is due to increased blood flow (hyperaemia) through the region, resulting in vascular dilatation and the delivery of warm blood to the area. Systemic fever, which results from some of the chemical mediators of inflammation, also contributes to the local temperature.

Swelling (tumor)

Swelling results from oedema, the accumulation of fluid in the extra vascular space as part of the fluid exudate, and to a much lesser extent, from the physical mass of the inflammatory cells migrating into the area.

Pain (dolor)

For the patient, pain is one of the best known features of acute inflammation. It results partly from the stretching and distortion of tissues due to inflammatory oedema and, in particular, from pus under pressure in an abscess cavity. Some of the chemical mediators of acute inflammation, including bradykinin, the prostaglandins and serotonin, are known to induce pain.

Loss of function (functio laesa)

Loss of function, a well-known consequence of inflammation, was added by Virchow (1821-1902) to the list of features drawn up by Celsus. Movement of an inflamed area is consciously and reflexly inhibited by pain, while severe swelling may physically

Early Stages of Acute Inflammation

1.Small blood vessels adjacent to the area of tissue damage initially become dilated with increased blood flow, then flow along them slows down.
2.Endothelial cells swell and partially retract so that they no longer form a completely intact internal lining.
3.The vessels become leaky, permitting the passage of water, salts, and some small proteins from the plasma into the damaged area (exudation). One of the main proteins to leak out is the small soluble molecule, fibrinogen.
4.Circulating neutrophil polymorphs initially adhere to the swollen endothelial cells (margination), then actively migrate through the vessel basement membrane (emigration), passing into the area of tissue damage.
5.Later, small numbers of blood monocytes (macrophages) migrate in a similar way, as do Iymphocytes.

Clinical indications of acute inflammation

General malaise

Fever

Pain, often localized to the inflamed area, e.g. the right iliac fossa in acute appendicitis

Rapid pulse rate

A raised neutrophil count in the peripheral blood.

An increased erythrocyte sedimentation rate (ESR).

An increase in the concentration of acute-phase proteins in the blood. These are normally present in small concentrations, but this increases dramatically in response to acute inflammation. Produced by the liver, they are induced by circulating IL-1. Specific examples, the most common being C-reactive protein, may be measured in blood to monitor inflammatory processes.

Le quattro cause secondo Aristotele

CAUSA MATERIALIS

CAUSA FORMALIS

CAUSA EFFICIENS

CAUSA FINALIS

Causes of Acute Inflammation

Microbial infections

Hypersensitivity reactions

Physical agents

Irritant and corrosive chemicals

.

Tissue necrosis

Systemic effects of acute inflammation

Pyrexia

Constitutional symptoms

malaise, anorexia and nausea.

Reactive hyperplasia of the reticulo-endothelial system

Haematological changes

Increased erythrocyte sedimentation rate..
Leukocytosis. Anaemia..

Le differenti associazioni di

calor, dolor, tumor, rubor e functio laesa producono

differenti quadri clinico-patologici

Macroscopic appearance of acute inflammation

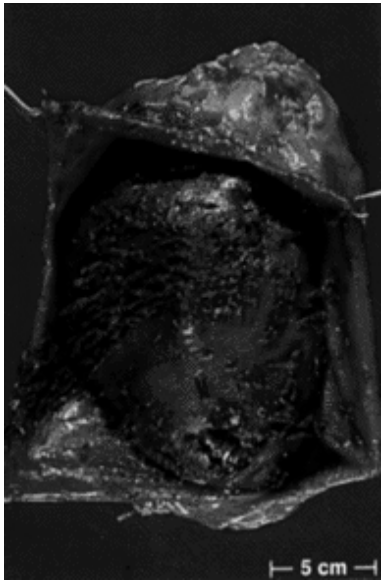
Serous inflammation

In serous inflammation, there is abundant protein-rich fluid exudate with a relatively low cellular content. Examples include inflammation of the serous cavities, such as peritonitis, and inflammation of a synovial joint, acute synovitis. Vascular dilatation may be apparent to the naked eye, the serous surfaces appearing injected, i.e. having dilated, blood-laden vessels on the surface, (like the appearance of the conjunctiva in 'blood- shot' eyes).

Catarrhal inflammation

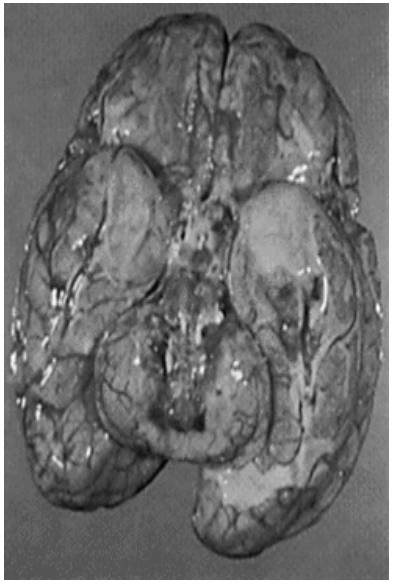
When mucus hypersecretion accompanies acute inflammation of a mucous membrane, the appearance is described as catarrhal. The common cold is a good example.

Fibrinous inflammation

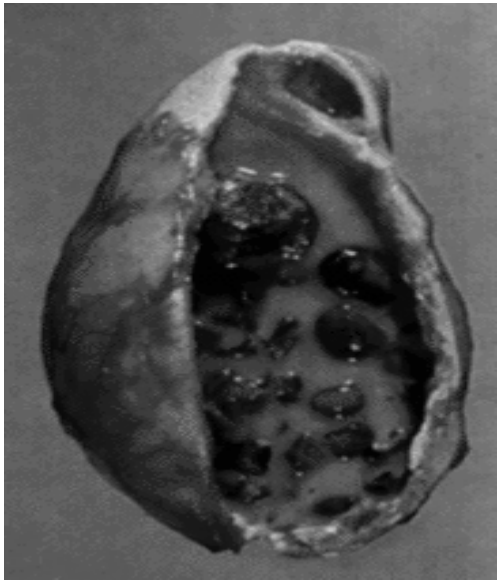


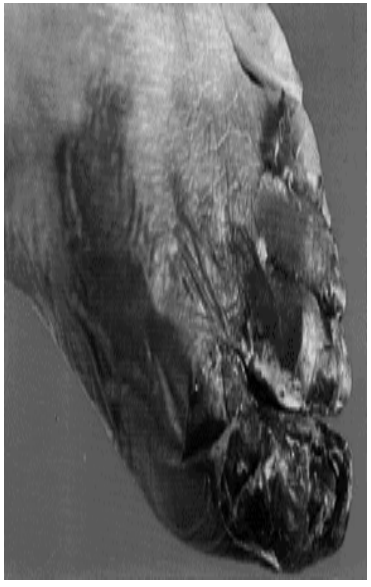
When the inflammatory exudate contains plentiful fibrinogen, this polymerises into a thick fibrin coating. This is often seen in acute pericarditis and gives the parietal and visceral pericardium a 'bread and butter' appearance.

Haemorrhagic inflammation



Haemorrhagic inflammation indicates severe vascular injury or depletion of coagulation factors. This occurs in acute pancreatitis due to proteolytic destruction of vascular walls, and in meningococcal septicaemia due to disseminated intravascular coagulation.





Suppurative (purulent) inflammation

The terms 'suppurative' and 'purulent' denote the production of pus, which consists of dying and degenerate neutrophils, infecting organisms and liquefied tissues. The pus may become walled-off by granulation tissue or fibrous tissue to produce an abscess (a localised collection of pus in a tissue) . If a hollow viscus fills with pus, this is called an empyema, for example, empyema of the gall bladder or of the appendix

Membranous inflammation

In acute membranous inflammation, an epithelium becomes coated by fibrin, desquamated epithelial cells and inflammatory cells. An example is the grey membrane seen in pharyngitis or laryngitis due to *Corynebacterium diphtheriae.*

Pseudomembranous inflammation

The term 'pseudomembranous' describes superficial mucosal ulceration with an overlying slough of disrupted mucosa, fibrin, mucus and inflammatory cells. This is seen in pseudomembranous colitis due to *Clostridium difficile* colonisation of the bowel, usually following broad-spectrum antibiotic treatment.

Necrotising (gangrenous) inflammation

High tissue pressure due to oedema may lead to vascular occlusion and thrombosis, which may result in widespread septic necrosis of the organ. The combination of necrosis and bacterial putrefaction is gangrene. Gangrenous appendicitis is a good example.

Effects of Acute Inflammation

Beneficial effect

Dilution of toxins.

Entry of antibodies.

Drug transport.

Fibrin formation.

Delivery of nutrients and oxygen.

Stimulation of immune response.

Harmful effects

Digestion of normal tissues.

Swelling.

Inappropriate inflammatory response.

Sequelae of Acute Inflammation

Resolution

Suppuration

Organisation

Chronic Inflammation

Resolution of Acute Inflammation

The term resolution means the complete restoration of the tissues to normal after an episode of acute inflammation. The conditions which favour resolution are:

minimal cell death and tissue damage

occurrence in an organ or tissue which has regenerative capacity (e.g. the liver)

rather than in one which cannot regenerate (e.g. the central nervous system)

rapid destruction of the causal agent (e.g. phagocytosis of bacteria)

rapid removal of fluid and debris by good local vascular drainage.

(e.g.Acute Lobar Pneumonia)

Suppuration

Suppuration is the formation of pus, a mixture of living, dying and dead neutrophils and bacteria, cellular debris and sometimes globules of lipid. The causative stimulus must be fairly persistent and is virtually always an infective agent, usually pyogenic bacteria (e.g. *Staphylococcus aureus*, Streptococcus pyogenes, Neisseria species or coliform organisms). Once pus begins to accumulate in a tissue, it becomes surrounded by a 'pyogenic membrane' consisting of sprouting capillaries, neutrophils and occasional fibroblasts. Such a collection of pus is called an abscess, and bacteria within the abscess cavity are relatively inaccessible to antibodies and to antibiotic drugs (thus, for example, acute osteomyelitis, an abscess in the bone marrow cavity, is notoriously difficult to treat).

Abscess

An abscess (for example, a boil) usually 'points', then bursts; the abscess cavity collapses and is obliterated by organisation and fibrosis, leaving a small scar. Sometimes, surgical incision and drainage is necessary to eliminate the abscess. If an abscess forms inside a hollow viscus (e.g. the gall bladder) the mucosal layers of the outflow tract of the viscus may become fused together by fibrin, resulting in an empyema.

Such deep-seated abscesses sometimes discharge their pus along a sinus tract (an abnormal connection, lined by granulation tissue, between the abscess and the skin or a mucosal surface). If this results in an abnormal passage connecting two mucosal surfaces or one mucosal surface to the skin surface, it is referred to as a fistula. Sinuses occur particularly when foreign body materials are present, which are indigestible by macrophages and which favour continuing suppuration. The only treatment for this type of condition is surgical elimination of the foreign body material.

Organisation

Organisation of tissues is their replacement by granulation tissue. The circumstances favouring this outcome are when:

Iarge amounts of fibrin are formed, which cannot be removed completely by fibrinolytic enzymes from the plasma or from neutrophil polymorphs

substantial volumes of tissue become necrotic or if the dead tissue (e.g. fibrous tissue) is not easily digested

exudate and debris cannot be removed or discharged.

During organisation, new capillaries grow into the inert material (inflammatory exudate), macrophages migrate into the zone and fibroblasts proliferate, resulting in fibrosis. A good example of this is seen in the pleural space following acute lobar pneumonia. Resolution usually occurs in the lung parenchyma, but very extensive fibrinous exudate fills the pleural cavity. The fibrin is not easily removed and consequently capillaries grow into the fibrin, accompanied by macrophages and fibroblasts (the exudate becomes 'organised'). Eventually, fibrous adhesion occurs between the parietal and visceral pleura.

Chronic Inflammation

Chronic inflammation is an inflammatory response of prolonged duration - weeks, months, or even indefinitely - whose extended time course is provoked by persistance of the causative stimulus to inflammation in the tissue. The inflammatory process **inevitably causes tissue damage** and is accompanied by simultaneous attempts at *healing and repair.* The exact nature, extent and time course of chronic inflammation is variable, and depends on a balance between the causative agent and the attempts of the body to remove it.

Chronic inflammation may develop in the following ways:

1)as a progression from *acute inflammation* if the original stimulus persists,

2)after repeated episodes of acute inflammation,

3) de novo if the causative agent produces only a mild acute response.

Aetiological agents producing chronic inflammation include:

Infectious organisms that can *avoid* or *resist* host defences and so persist in the tissue for a prolonged period. Examples include Mycobacterium tuberculosis, Actinomycetes, and numerous fungi, protozoa and metazoal parasites. Such organisms are in general able to avoi phagocytosis or survive within phagocytic cells, and tend not to produce toxins causing acut tissue damage.

1)Infectious organisms that are not innately resistant but persist in damaged regions where they are protected from host defences. The common example here is of bacteria which grow i the *pus within an undrained abscess cavity*, where they are protected both from host immunit and from blood-borne theraputic agents, e.g. antibiotics. Some locations are particularly pron to chronic abscess formation, e.g. bone, pleural cavities.

2)Irritant non-living foreign material that cannot be removed by enzymic breakdown or phagocytosis. Examples include a wide range of materials implanted into wounds (wood splinters, grit, metals and plastics), inhaled (silica dust and other particles or fibres), or deliberately introduced (surgical prostheses, sutures, etc.). Dead tissue components that cann be broken down may have similar effects, e.g. keratin squames from a ruptured epidermoid cyst or fragments of dead bone (sequestrum) in osteomyelitis.

3)In some cases the stimulus to chronic inflammation may be a "normal" tissue component. This occurs in inflammatory diseases where the disease process is initated and maintained because of an abnormality in the regulation of the body's immune reponse to its own tissues the so-called *auto-immune diseases.*

4)For many diseases characterised by a chronic inflammatory pathological process the underlying cause remains unknown. A good example here is Crohn's disease of the intestine

Histological appearances in chronic inflammation:

The microscopic appearances of chronic inflammation vary considerably according to the site involved and the causative stimulus. However, the general features are:

A mixed inflammatory cell infiltrate containing predominantly macrophages, lymphocytes and lasma cells, with neutrophil and eosinophil polymorphs as possible minor components (compar with the predominance of *neutrophils* in acute inflammation). Lymphoid cells can proliferate a the site of inflammation as well as in local lymph nodes; in severe cases this can give rise to lymphoid follicles with germinal centres in the inflammatory lesion.

Tissue destruction (*necrosis*) caused both by the causative agent and by the inflammatory proces itself.

Attempts at reconstructing the damaged tissue occur simultaneously with the inflammatory process. These can be considered under the general title of healing and repair. The attempts at reconstruction may have different outcomes. If there is little tissue destruction then some organ may be able to regenerate their original structure, or mild inflammation may terminate by resolution without causing any structural damage. Commonly, however, the original structure cannot be re-created and the damaged area undergoes repair.This involves removal of the estroyed tissue by phagocytosis with proliferation of capillary blood vessels and lymphatics int he lesion together with fibroblasts and collagen production (so-called granulation tissue), endin up with a dense fibrous *scar*.

Granulomatous inflammation is a histologically distinctive form of chronic inflammation tha ccurs in particular circumstances in response to certain organisms or foreign material and meri escription in a separate section. **N.B.** This term (granuloma, granulomatous inflammation) is **n to be confused** with granulation tissue. Look at the links explaining both processes and be sure you understand the distinction

Mechanisms of control of the chronic inflammatory response:

Accumulation of macrophages and lymphocytes in areas of chronic inflammation occurs in at least three ways:

1)Continued recruitment from the circulation.

2)Local proliferation.

3)Prolonged survival and immobilisation in the inflamed area.

Control of the development, maintenance and termination of inflammation is performed through *chemical mediators* released from damaged tissue, from the inflammatory cells themselves, and from enzyme systems in blood plasma. The healing and repair processes are also *controlled by chemical interactions* between the cells and extracellular tissue elements involved.

Chemical Mediators of Acute Inflammation

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Chemical mediators released from cells

Histamine. This is the best-known chemical mediator in acute inflammation. It causes vascular dilatation and the immediate transient phase of increased vascular permeability. It is stored in mast cells, basophil and eosinophil leukocytes, and platelets. Histamine release from those sites (for example, mast cell degranulation) is stimulated by complement components C3a and C5a, and by Iysosomal proteins released from neutrophils.

Lysosomal compounds. These are released from neutrophils and include cationic proteins, which may increase vascular permeability, and neutral proteases, which may activate complement.

Prostaglandins. These are a group of long-chain fatty acids derived from arachidonic acid and synthesised by many cell types. Some prostaglandins potentiate the increase in vascular permeability caused by other compounds. Others include platelet aggregation (prostaglandin 1. is inhibitory while prostaglandin A2 is stimulatory). Part of the anti-inflammatory activity of drugs such as aspirin and the non-steroidal anti-inflammatory drugs is attributable to inhibition of one of the enzymes involved in prostaglandin synthesis.

Chemical mediators released from cells

Leukotrienes. These arc also synthesised from arachidonic acid, especially in neutrophils, and appear to have vasoactive properties. SRS-A (slow reacting substance of anaphylaxis), involved in type I hypersensitivity, is a mixture of leukotrienes.

5-hydroxytryptamine (serotonin). This is present in high concentration in mast cells and platelets. It is a potent vasoconstrictor.

Lymphokines. This family of chemical messengers released by Iymphocytes. Apart from their major role in type IV hypersensitivity, Iymphokines may also have vasoactive or chemotactic properties.

Plasma factors

Complement system. The complement system is a cascade system of enzymatic proteins. It can be activated during the acute inflammatory reaction in various ways:

In tissue necrosis, enzymes capable of activating complement are released from dying cells.

During infection, the formation of antigen-antibody complexes can activate complement via the classical pathway, while the endotoxins of Gram-negative bacteria activate complement via the alternative pathway.

Kinin system. The kinins are peptides of 9-11 amino acids; the most important vascular permeability factor is bradykinin. fibrinolytic systems.

Fibrinolytic system. Plasmin is responsible for the Iysis of fibrin into fibrin degradation products, which may have local effects on vascular The kinin system is activated by coagulation factor XII. Bradykinin is also a chemical mediator of the pain which is a cardinal feature of acute inflammation.

Coagulation system. The coagulation system is responsible for the conversion of soluble fibrinogen into fibrin, a major component of the acute inflammatory exudate.

Coagulation factor XII (the Hageman factor), once activated by contact with extracellular materials such as basal lamina, and various proteolytic enzymes of bacterial origin, can activate the coagulation, kinin and permeability

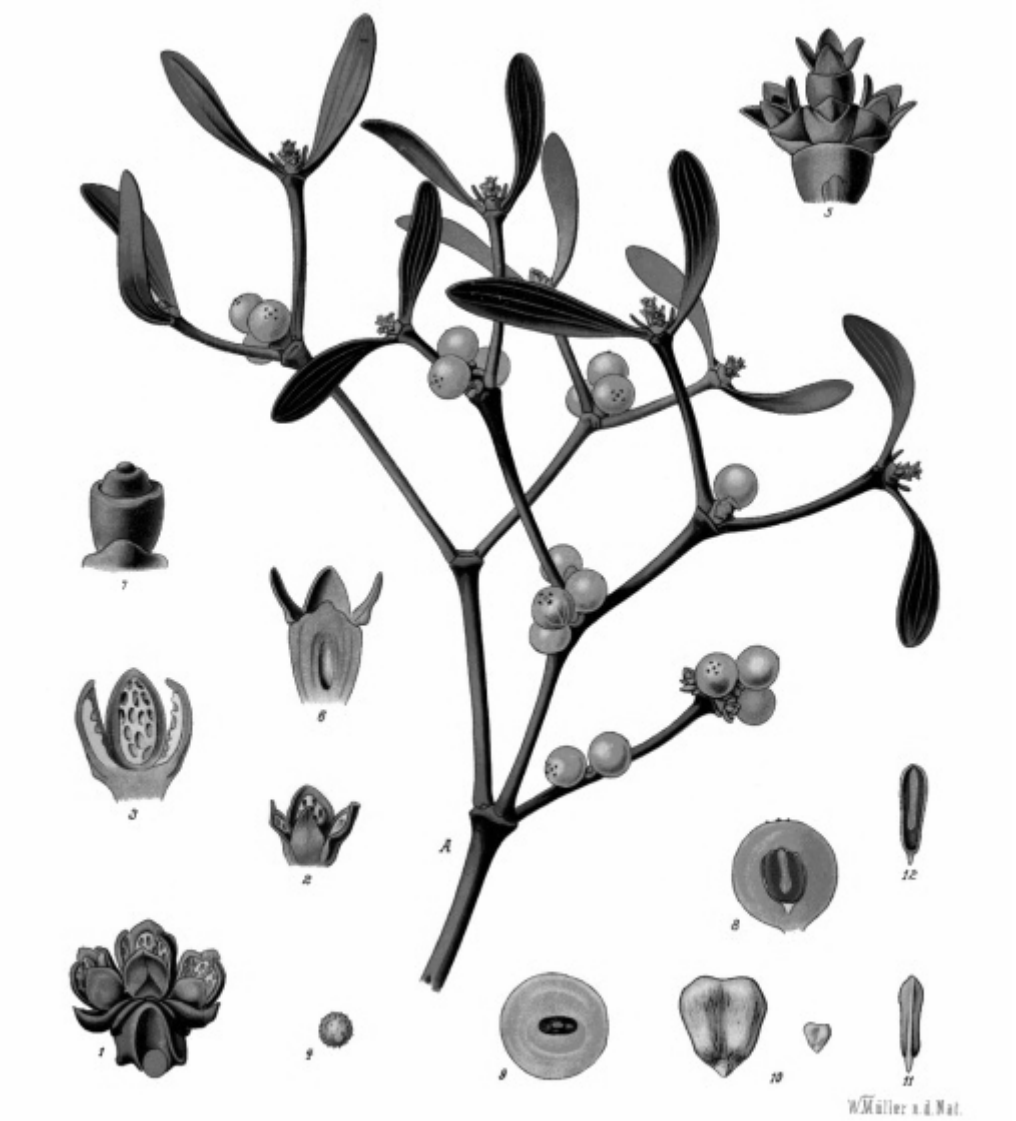
Spunti bibliografici sull'infiammazione nell'opera di Rudolf Steiner

1 "L'uomo invisibile in noi" (sul tema : sangue e nervo).

2)"Scienza dello spirito e medicina". Conff. XIII e XIV (sui temi: infiammazione e tumori;i fantomi della vista e dell'udito).

3) "Elementi fondamentali per un ampliamento dell'arte medica". Cap.X (sul tema:grassi e infiammazione).

4) "Le manifestazioni del Karma". Conf. IV (sul tema: malattie luciferiche e arimaniche)



Viscum album L.

Parametri diagnostici nell'ambito funzionale degli arti costitutivi
(Adattato da : M.Gloecker "Anthroposophische Arzneitherapie")

Arto costitutivo e leggi di natura	Esperienza e creatività	Morfofisiologia	Sintomi fisici
Organizzazione dell'io Organismo calorico Leggi termodinamiche.	Attività pensante autocosciente Intenzionalità ,Iniziativa, Poesia Arte della Parola.	Processi integrativi, Forma complessiva.	Ripartizione individuale del calore, "Irradiazione" CALOR
Corpo astrale Organismo aeriforme Leggi aerodinamiche.	Coscienza,Sentimento,Impulsività, Movimento,Musica, Canto.	Respirazione Catabolismo Eterostasi Differenziazione Proporzioni.	Tono muscolare, Gestione dell'aria. DOLOR
Corpo eterico Organismo acqueo Leggi idrodinamiche	Spinta archimedea Levità, Cronobiologia, Processi plastici.	Anabolismo Sintesi Omeostasi Proliferazione	Turgore, Gestione dell'acqua,Colorito, Sanguificazione cutanea TUMOR,RUBOR
Corpo fisico Organismo solido Gravità Meccanica	Gravità Esperienza architettonica	Processi di deposito e strutturazione,Mantenimento della forma.	Spessore,Peso, Parametri laboratoristici FUNCTIO LAESA

INFIAMMAZIONE

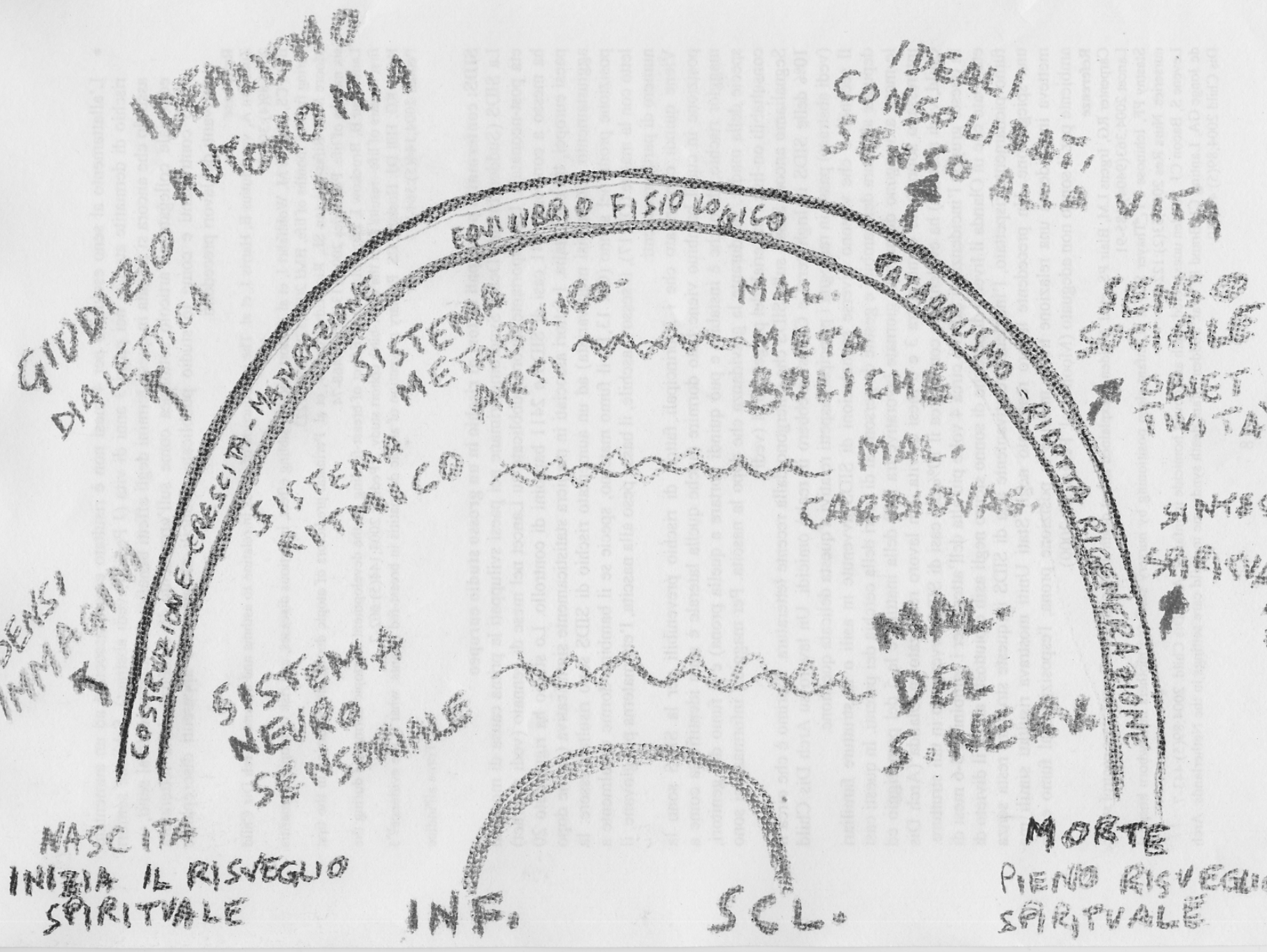
- SANGUE
- CENTRIFUGA
- CALORE
- DISSOLUZIONE
- OCCHIO
- ISTERIA
- FEMMINILE
- GIOVINEZZA
- ETC.

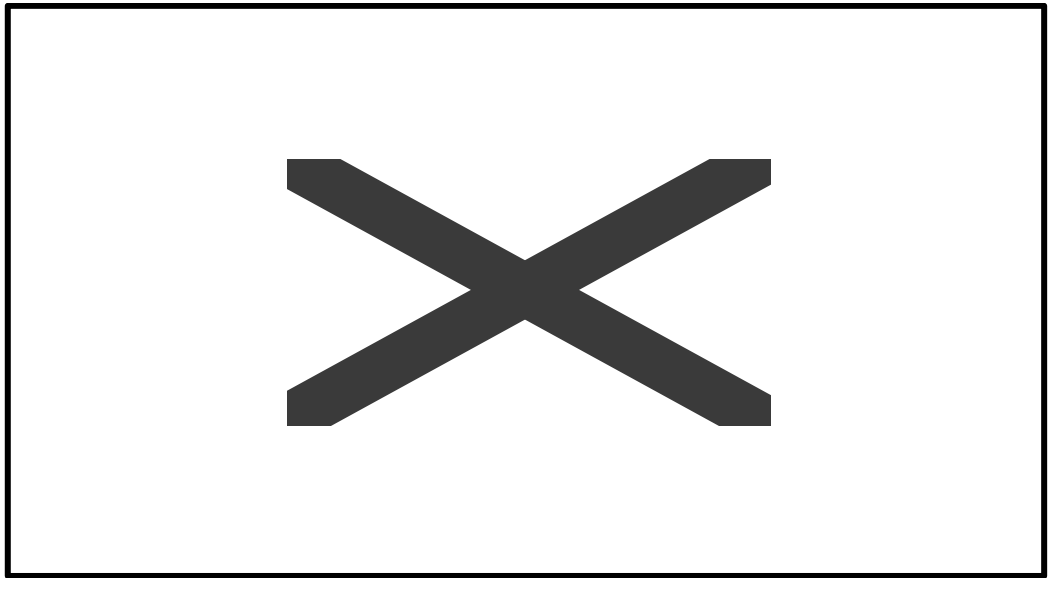
LUCIFERO

ARIMANE

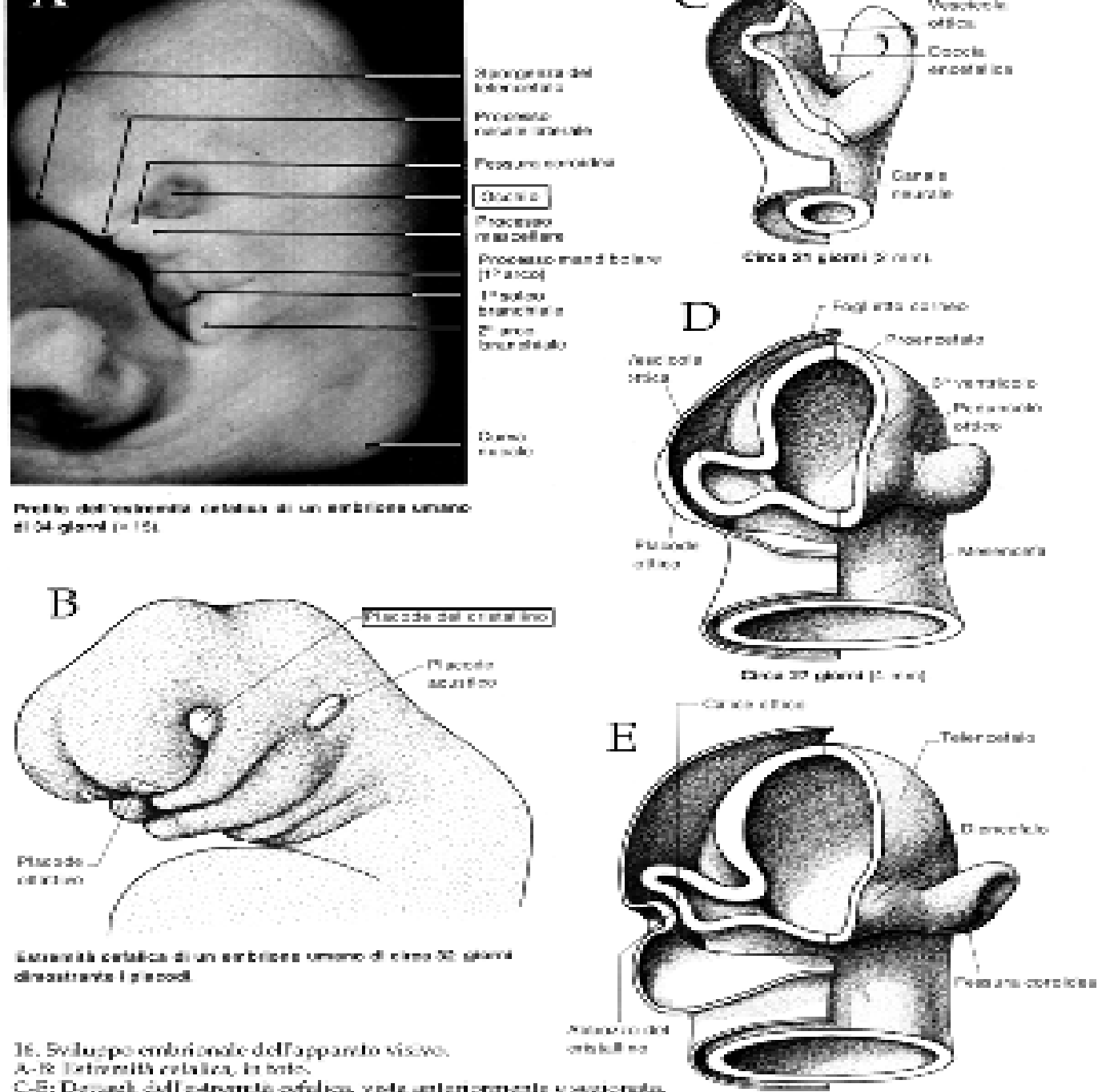
SCLEROSI

- NERVO
- CENTRIPETA
- RAFFREDDAMENTO
- INDURIMENTO
- ORECCHIO
- NEVRASTENIA
- MASCHILE
- VECCHIAIA
- ETC.


IDENTISMO
AUTONOMIA
IDEALI
CONSOLIDATI
SENSO ALLA VITA
GIUDIZIO
DIALETTICA
EQUILIBRIO FISIOLOGICO
COSTRUZIONE - CRESCITA - MANTENIMENTO
CATABOLISMO - RIPARAZIONE - RIGENERAZIONE
SISTEMA METABOLICO
MAL. METABOLICHE
SENSO SOCIALE
OBIETTIVI
SISTEMA RITMICO
MAL. CARDIOVAS.
SENSI IMMAGINI
SISTEMA NEURO SENSORIALE
MAL. DEL S. NERV.
NASCITA
INIZIA IL RISVEGLIO SPIRITUALE
INF.
SCL.
MORTE
PIENO RISVEGLIO SPIRITUALE



“In ogni caso di vera infiammazione…il corpo eterico umano
è attivo nella sua totalità” (Scienza dello spirito e medicina, conf.XIII)
“Un’eccessiva intensità della formazione di quell’impalcatura (che nel corpo fisico somiglia a quello eterico) fornisce l’occasione a processi infiammatori e alle loro possibili conseguenze…Quell’impalcatura tende allo sfacelo infiammatorio solo se non è energicamente compenetrata dall’io…Si possono congiungere grazie all’uso dell’acido formico aggiunto all’acqua del bagno in alta diluizione, perché in questo modo le forze dell’acido formico vengono potenziate” (Scienza dello spirito e medicina conf.XIV)



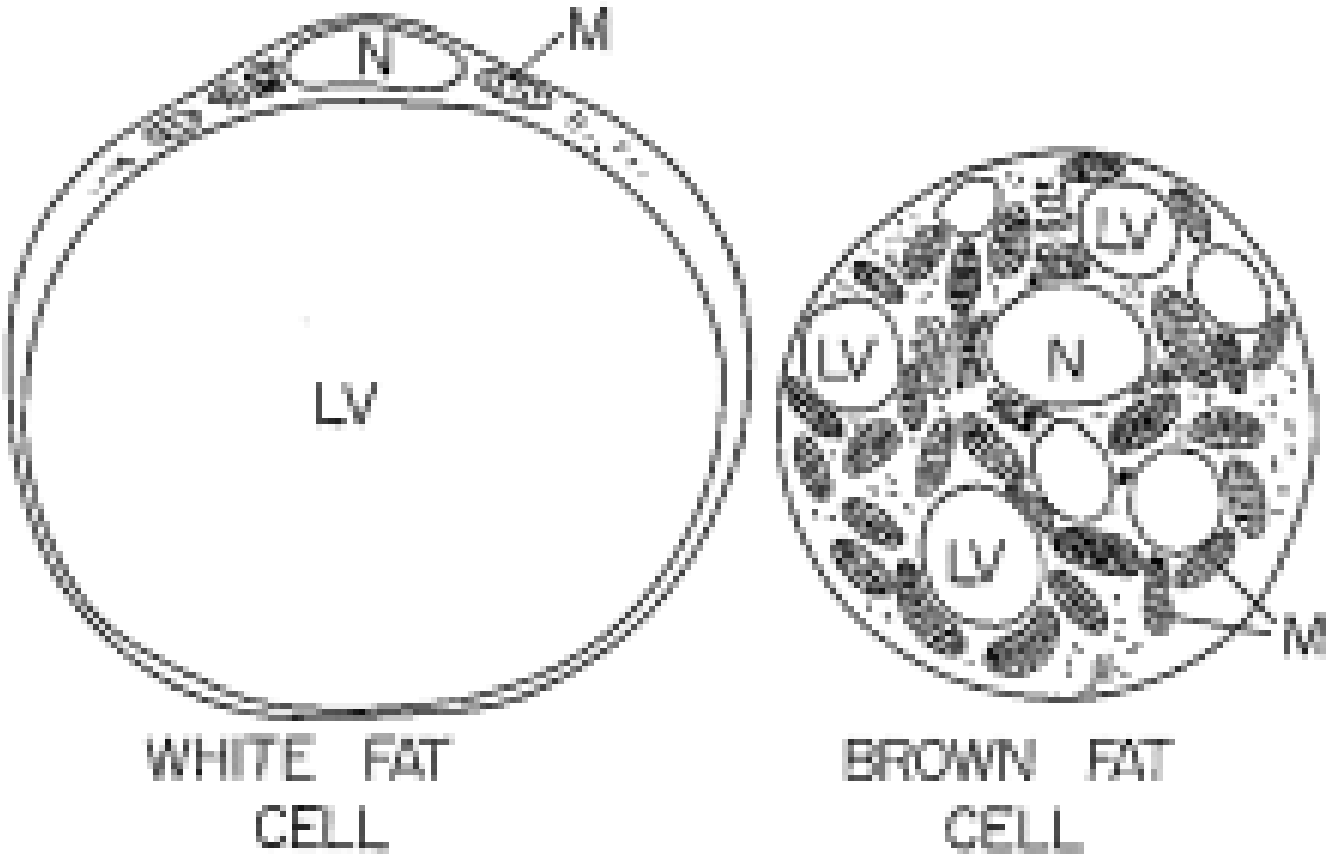
16. Sviluppo embrionale dell'apparato visivo.
A-B: Estremità cefalica, in toto.
C-E: Passaggi dell'estremità cefalica, viste anteriormente e ventralmente.

“L’acido silicico ha un duplice compito : esso pone un limite interno ai processi di crescita, nutrizione,etc. Inoltre separa, verso l’esterno, le azioni della natura esterna dall’interno dell’organismo.” (Steiner-Wegman)

Eccesso di processo del silicio verso la periferia-Sonnolenza, vertigini come se ristagnasse in periferia la conduzione dell’impulso nervoso.

Eccesso del processo del silicio verso l’interno- dolori articolari, infiammazioni, dove troppo s’inserisce l’attività portatrice di forma del silicio stesso

Bisogna osservare se i fenomeni infiammatori si manifestano in soggetti con tendenza all'obesità.
Infatti il trattamento esterno,cioè la menzionata applicazione dell'acido formico,di origine animale, può dare buoni risultati solo se si trovano associati i due complessi sintomatici.
(Scienza dello spirito e medicina , conf. XIV)



WHITE FAT CELL

BROWN FAT CELL

acido arachidonico

Nome IUPAC	
acido *cis,cis,cis,cis*-5,8,11,14-icosatetrenoico	
Caratteristiche generali	
Formula bruta o molecolare	$C_{20}H_{32}O_2$
Massa molecolare (amu)	304,48
Aspetto	liquido incolore
Numero CAS	506-32-1
Proprietà chimico-fisiche	
Densità ($g \cdot cm^{-3}$, in c.n.)	0,922
Indice di rifrazione	1,4870
Solubilità in acqua	insolubile
Temperatura di fusione (K)	224 (-49°C)

Quando mi vide star pur fermo e duro,
turbato un poco disse: «Or vedi, figlio:
tra Beatrice e te è questo muro».
Come al nome di Tisbe aperse il ciglio
Piramo in su la morte, e riguardolla,
allor che 'l gelso diventò vermiglio;
così, la mia durezza fatta solla,
mi volsi al savio duca, udendo il nome
che ne la mente sempre mi rampolla.
Ond'ei crollò la fronte e disse: «Come!
volenci star di qua?»; indi sorrise
come al fanciul si fa ch'è vinto al pome.
Poi dentro al foco innanzi mi si mise,
pregando Stazio che venisse retro,
che pria per lunga strada ci divise.
Sì com'fui dentro, in un bogliente vetro
gittato mi sarei per rinfrescarmi,
tant'era ivi lo 'ncendio sanza metro.
Lo dolce padre mio, per confortarmi,
pur di Beatrice ragionando andava,
dicendo: «Li occhi suoi già veder
parmi».