

V - ALTERATIONS IN BODY DEFENSES

V.A. STRESS & ADAPTATION

A.a. GENERAL

Even when diseased (stressed), many body functions go on as normal, a fact worth pointing out to your client.

A.b. STRESS

A.b.A. GENERAL PATHOLOGY

stress { } > <disease> (hypertension, heart disease, peptic ulcer)
 { } > behaviors (smoking, alcohol abuse, overeating) => <disease>

Path VA-1. General Pathology of Stress

A.b.B. CATEGORIES OF STRESS

A.b.B.a. PHYSIOLOGICAL~ADAPTIVE~EUSTRESS

- often
- 1) highly predictable
- 2) specific
- 3) turns off when no longer needed (e.g. \uparrow temp \rightarrow \uparrow HBR)

A.b.B.b. PSYCHOLOGICAL~MALADAPTIVE~DISTRESS

- 1) not predictable
 - 2) nonspecific
- (Hans Selye, endocrinologist): ["Nonspecific response of the body to any demand made on it."]; nonspecific [logically inappropriate, nonadaptive]
- 3) continues beyond removal of stress

A.b.C. STRESSORS [sources of stress] (Δ in stressor {intensity or type} => Δ demand on body => stress response)

A.b.C.a. CLASSIFICATION

- 1) according to the source of stressors

Endogenous arise w/in the body
Exogenous arise outside the body

- 2) according to the timing/sequencing of stressors (1,2,3, below, where each different number represents a different stressor)

<u>Acute Time-Limiting</u>	1		
<u>Event-Sequencing</u>	1	2	3
<u>Chronic Intermittent</u>	1	1	1
<u>Chronic Sustained</u>	1	1	1

A.b.D. GENERAL ADAPTATION SYNDROME (GAS)

Adaptation [responses to changing conditions likely to promote survival]

- 1) Three stages

- 1) Alarm Stage: \uparrow hypothalamus-pituitary-adrenal (HPA) axis \rightarrow mobilization of energy & \uparrow efficiency of circulatory system \rightarrow Selye's triad: adrenal enlargement, thymus atrophy & gastric ulcers
- 2) Stage of Resistance: \downarrow HPA Axis + \uparrow system best able to deal w/ stressor
- 3) Stage of Exhaustion: all systems worn out, especially those in stage 2

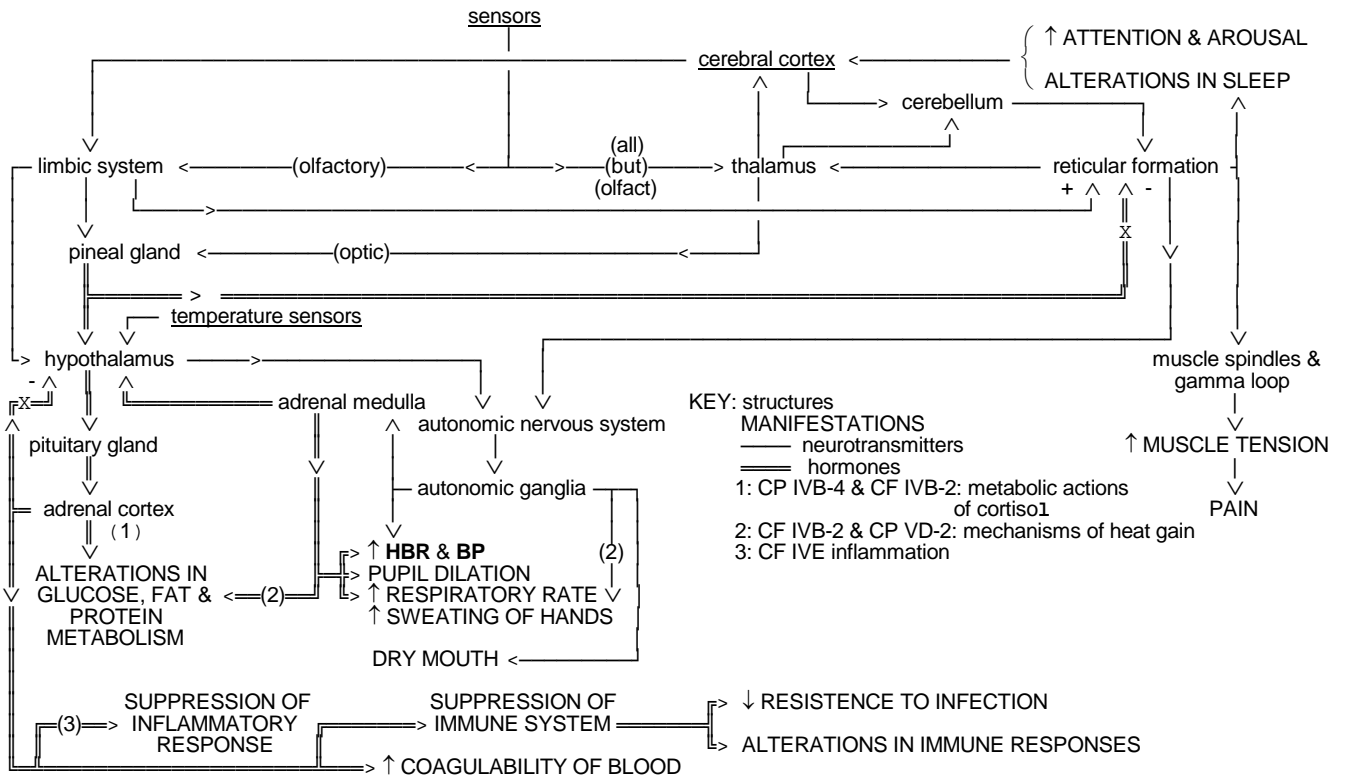
2) In first two stages, homeostatic responses [responses \rightarrow a constant condition for some physiological process in the body] commonly occur. These responses can regulate some physiological aspect \uparrow or \downarrow ; each homeostatic response mechanism has the following four components:

<u>physiological aspect</u>	<u>sensor</u>	<u>comparator</u>	<u>effector system</u>
<u>variable</u> that is <u>measured</u> = ?	<u>measures value</u> of <u>variable</u> = ?	compares <u>measured value</u> to an established <u>set point</u>	some body system that brings <u>measured value</u> to <u>set point</u>
()	()	()	()

HWA

Figure VA-1. The Four Components of a Homeostatic Response TP VA-1

A.b.E. RESPONSES TO (MANIFESTATIONS OF) STRESS

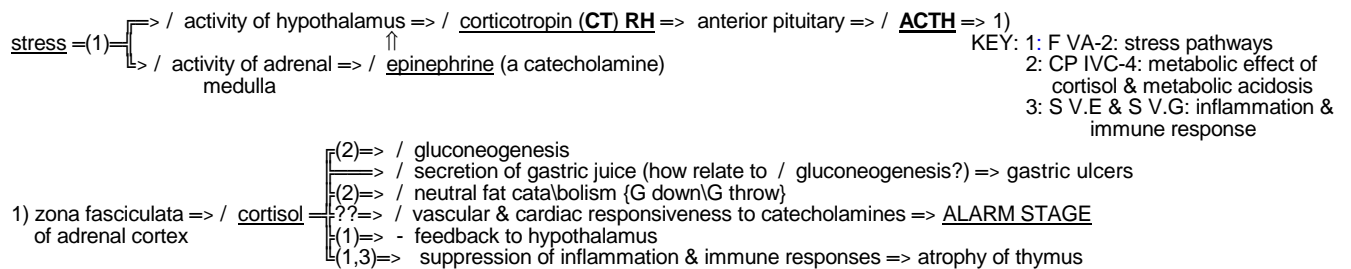


Critical Figure VA-2. Stress Pathways. =X=> represents negative feedback-inhibitory effect; adopted from 32, p 147) TP VA-2 HWA

1) Auto\onomic {G self\G law} Responses

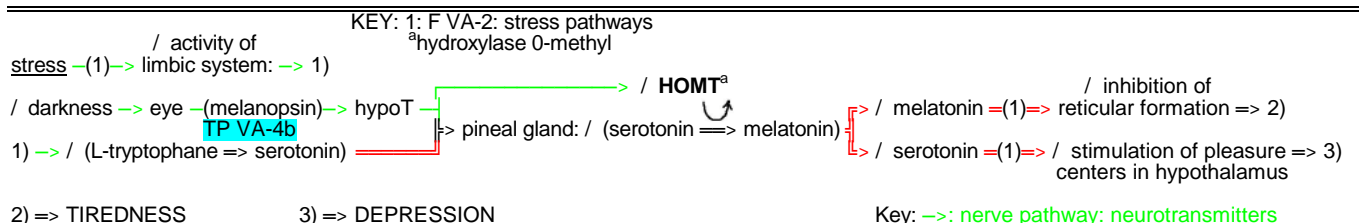
- ↓ para\sympathetic {G along side of\} division (routine homeostatic)
- ↑ sympa\thetic {G together\G squeeze\} division (emergency homeostatic):
 - 1) activate emergency survival mechanisms (pupils dilate, ↑ heart BR, ↑ respiratory rate)
 - 2) partition limited supply of blood to where it will do most good
 - 3) Table 21.1, p 591, TP VA-3
 - 4) involves HPA Axis

2) Hypothalamic-Pituitary-Adrenal Responses (Axis)



Path VA-2. Path from Stress Through the HPA Axis to the Alarm Stage (9, → p 260) TP VA-4

3) Limbic System-Pineal Gland-Hypothalamic Axis



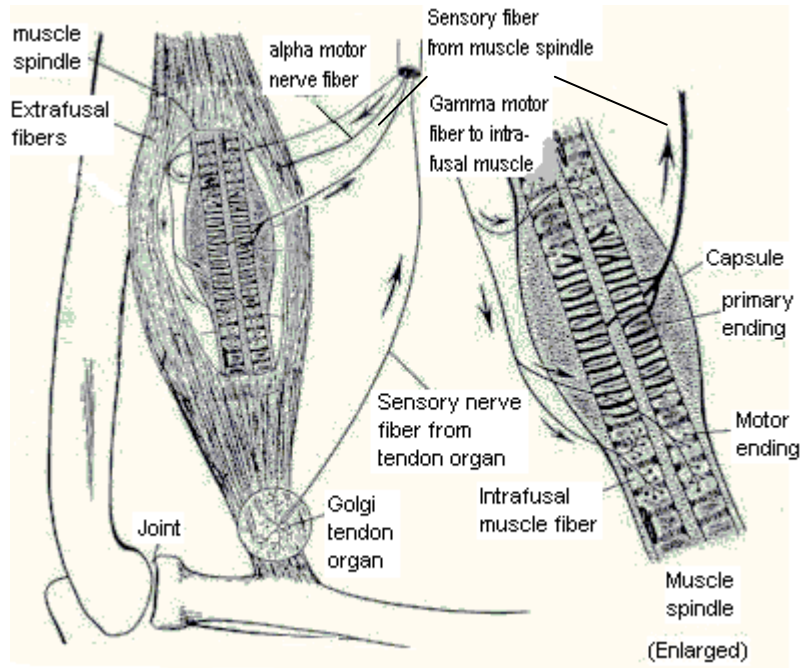
Path VA-3. Path from Stress Through the Pineal Gland to the Hypothalamus TP VA-4a

4) Musculoskeletal Responses (musculoskeletal tension = stiff neck, backache, headache, knee ache)

KEY:

A = α -motor nerve fiber- α -motoneuron
 B = extrafusil muscle fiber
 {G outside of G spindle}
 C = stretch sensor in muscle spindle
 D = sensory fiber from muscle spindle
 E = γ -motor nerve fiber- γ -motoneuron
 F = intrafusil muscle fiber
 {G within}

A -> B
 C -> D
 E -> F



Critical Figure VA-3. The Structure of Muscle Spindles & Their Relationship to Muscle Fibers (3, p 186) **TP VA-5**

TP VA-6

resting muscle: F is always contracted > B, so C is always actively stretched

contracting muscle: \uparrow stimulation of A \rightarrow / contraction of B \rightarrow / stretch of C \rightarrow / impulses in D \rightarrow / impulses on E \rightarrow / contraction of F \rightarrow 1)
 (shortening of muscle)

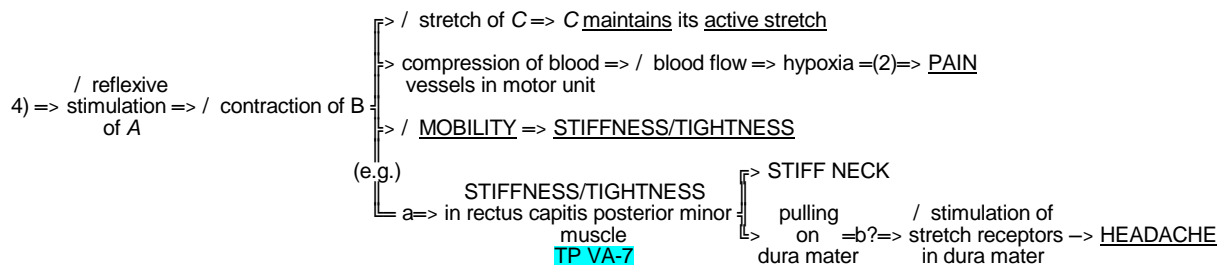
1) \rightarrow / stretch of C so C maintains its active stretch status

relaxing muscle: (\downarrow stimulation of A \rightarrow) / contraction of B \rightarrow / stretch of C \rightarrow / impulses in D \rightarrow 2)
 (or muscle being stretched by contraction of antagonist) (elongation of muscle)

2) \rightarrow / impulses on E \rightarrow / contraction of F \rightarrow so C maintains its active stretch

stress = (1) \Rightarrow / stimulation of (SO) cerebral cortex = (1) \Rightarrow / SO limbic system = (1) \Rightarrow / SO reticular formation \Rightarrow / impulses on E \Rightarrow 3)

3) \Rightarrow / contraction of F \Rightarrow too much (/) stretching of C \Rightarrow / impulses in D \Rightarrow 4)



KEY: 1: F VA-2: stress pathways

2: CP IIIA-9: severe hypoxia \rightarrow / lactic acid \rightarrow / stimulation of pain receptors

a: (41, pp 2484-2485)

b: (42, p 2486)

Critical Path VA-4. Path From Stress to Pain & Stiffness

5) Immunologic & Hematologic Responses (S V.F & V.G: white blood cells & immunology; S V.I: hemostasis)

6) Blood-brain Barrier Responses (43, p 375) involves CRH, the immune system, cytokines (IL-1) and nerve cell death **(BIODOTS)**