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UNITED KINGDOM

Dear Jim:

For several months now, I've been wanting to get your reaction to some ideas regarding a quantitative statement of a conflict theory for motion sickness, and report to you my experiences using vision restricting glasses while weightless on NASA's KC-135, as well as some experiments we've started here on subjective scaling of motion sickness symptomatology. I've been meaning to write you a proper letter for some time, and have been feeling quite guilty that the press of spring term teaching and Spacelab crew training has kept me from it.

Let me start with some modelling notions. I came away from our discussions at Aspen convinced it might be productive to try to develop a mathematically concise statement of the conflict theory which could both capture the major themes of your '78 Neural Mismatch model, and also fit easily with the spatial orientation modelling work we've been doing here for some years. Such a model would at least be valuable for heuristic purposes, so that when planning experiments and interpreting data, the hypothesis is very concisely stated. And it might even be made tractable enough to permit numerical simulation via computer. I started this effort last summer, and presented preliminary results informally at a vestibular meeting at NASA's Johnson Space Center in the late fall. I very much would like to solicit your suggestions and criticisms as I start writing it up; there may well be some fundamental considerations which I have overlooked.

In trying to assemble a model, I was immediately faced with several issues, some of which we have already discussed: How to represent a neural trace or engram analytically? Temporally, does it have a beginning and an end? Or is it really appropriate to think of the neural store as a dictionary containing sequences of matched motor command and reafference time histories? Are there other functionally equivalent but more tractable ways to represent the Neural Store? Could it be, for example, that the Neural Store really only contains the information (i.e. the decision rules) necessary to generate motor and efferent copy sequences given a motor command, and not the sequences themselves? Also, existing statements of the conflict theory have relatively little specific to say about the normal, functional role of sensory conflict information, other than triggering updating of the neural store, and to contribute somehow to illusions and reflex reactions. What can be

said about the need for a conflict signal from the point of view of control theory? With all these neural circuits presumably present, why is the CNS so interested in sensory conflict? Other issues also arise: Mustn't one postulate an additional dynamic lag between the generation of a conflict signal and the appearance of symptoms? Do we really know that there is a threshold phenomenon associated with motion sickness at the level of the comparator element in your Neural Mismatch model, and not just at the back end of the model, where symptoms appear?

In order to deal with all this, I decided to take a look at the problem of how the CNS might achieve a closed loop volitional and reflex control of body orientation from a theoretical point of view, using a mathematical approach developed in engineering in the late 1950s and early 60s. The theory deals specifically with the optimal control of complex systems in the special situation where feedback sensors don't provide complete information on what the controlled element (the body) is doing at any given moment in time. My rationale for this was that there ought to be a simple, theoretical explanation for why the CNS needs a Neural Store and a conflict signal to achieve control of body orientation. Control theory immediately suggested an answer: If the CNS is faced with the task of controlling body, limb, and head position using partially redundant, but incomplete, information about the body state from biologically noisy sensory organs, then an appropriate strategy (indeed, the optimal strategy under certain conditions) is for the central nervous system to employ internal models of the body and its sensory systems to predict, from moment to moment, an internal estimate of what the body is doing. This estimate is presumably associated with perceived orientation. The internal models are also used to predict the sensory input to be expected under conditions where the body is not subjected to externally imposed movement; if the sensory systems are noise free; and if the internal models are correct. Since, of course, these conditions are not normally met in engineering or living systems (if they were, all movements could be controlled in an open loop fashion using only engrams generated using the internal models), the theory says that the appropriate approach is to compare the actual sensory afference with the model generated predicted sensory input, and specifically says how the conflict signal should be used to "update" the predictions made by the internal models. The theory also provides a convenient alternative method for dealing with the concept of an engram or neural trace, as well as the dictionary concept of the Neural Store.

The theory itself is a variant of the theory of optimal linear output feedback control systems as developed by Kalman and, later, Wonham. Fortunately, the details of the theory aren't particularly important, and anyone who has ever learned how to multiply two matrices together can easily grasp the concept. The attached figures will give you the flavor of the basic arguments. Suppose, as shown in Figure 1, the job of the CNS is to stabilize body sway using only ankle joint, semicircular canal, and otolith information. (I've left vision out, but only for simplicity. No sacrilege intended.) Since it's notationally cumbersome to deal with whole sets of differential equations, one can simplify the problem notationally and conceptually by writing the equations in matrix form, as shown in Figure 2. To do this, you define a vector, X, whose components are the physical variables in the body and its sensors which are important in determining body and sense organ bahavior. For example, body sway angle and its rate of change, cupula deflection and its rate of change, otolith displacement, etc. You also define a forcing vector, U, which is made up of all the active and passive forces and torques acting on the body. When you do this, you find (Figure 2) that you can rewrite the complicated set of equations in the much simpler form $\dot{X} = AX + BU$.

You can easily visualize this equation by looking at the top of Figure 3: the actual state of the body and its sensors, X, is drawn as the output of an integrator, whose input, $\dot{X}$, is just the sum of two vectors AX and BU. The matrix A contains the coefficients of the differential equations describing the unforced (free; homogeneous solution) behavior of the body and sensors; the matrix B is made up of coefficients expressing body inertia, etc., and dictates how the rate of change of body and sensor state, $\dot{X}$, is influenced by active and passive forces. Sensory input to the CNS is calculated just by multiplying the "actual state " vector, X, by a matrix S which represents the static gains of the sensory organs. Many of the terms in the S matrix are likely zero, since the CNS isn't, in general, provided with information about all aspects of the state of the body.

If you more or less followed the last paragraph, the rest of the model is easy. As shown in Figure 4, the U vector is composed of two sorts of forces and torques: Unpredictable, externally imposed torques η_e, and torques resulting from active motor outflow to muscle, denoted m. Motor outflow, m, is assumed to result from a control strategy matrix C, whose input is an error signal: the difference between a desired state X_d (corresponding to the command signal in your latest model and the "will" in J.J. Groen's model for motion sickness) and an internally generated estimate of the actual body state $\hat{X}$. The notion of an internal state estimate is vital. Since the CNS cannot directly observe the true state of the body, X, it is forced to use an internal model (which corresponds to your Neural Store) to generate, from moment to moment, an estimate of the actual state. How this internal estimate is generated in the absence of any sensory information is shown in Figure 5. The internal model estimate $\hat{X}$ is generated by a vector integrator, whose input is the rate of change of the internal estimate, $\dot{\hat{X}}$. The critical issue is how the CNS might estimate $\dot{\hat{X}}$. Lacking sensory input, the CNS might take advantage of all its previous experience in controlling the body, and employ the information in a matrix transformation $\hat{A}$, which represents the CNS' best estimate of what the passive behavior characteristics of the body have been in the recent past, i.e. an estimate of the matrix A. Since the motor outflow to muscles is presumably available to the CNS, it presumably could also employ a matrix $\hat{B}$, the CNS' best estimate of the matrix B, to predict the expected effect of motor commands on $\dot{\hat{X}}$. In the absence of any sensory input, the CNS could achieve adequate open loop control of the body provided that it was working with the correct model (formally, that $\hat{A} = A$ and $\hat{B} = B$) and the body was not subject to any external disturbances η_e. If you were to give me the time history of the desired state X_d, the model would generate a continuous motor "engram". (Presumably, this reflects how Taub's monkeys achieve preprogrammed motor control when deafferented. I've discussed this modelling concept with Emilio Bizzi, and he agrees it is a good way to get around the problem of defining the beginning and end of an engram. It is important, though, to note that what is retained in the CNS internal model is not the engram itself, but rather the infromation needed to generate it, specifically the $\hat{A}$ and $\hat{B}$ matrices.)

Of course, humans are exposed to external forces, and their internal models for body and sensory system dynamics may be only approximate. This is true in engineering control systems as well. So the trick is to figure out a way to blend in information from sensory systems to update the internal model estimate of what the body is doing. I don't think that Rudy Kalman had ever heard of Von Holst or efferent copy. But Kalman did show that the best strategy is to adopt an internal estimate of the steady state sensitivities of the sensory systems, S, and multiply

this by the estimated state vector $\hat{X}$, as shown in Figure 6, to predict, from moment to moment, the expected sensory input. If the internal model is roughly correct, sensory system noise is low, and the body is not subject to external disturbances, then this efferent copy signal will closely approximate the actual sensory input. The sensory/motor conflict vector, c, equal to the difference between the actual and expected sensory input, will be small. But even if c is small, the system can still take advantage of the conflict signal to "tweak up" an improved estimate of body state. This is accomplished by multiplying the conflict vector c by a matrix K whose coefficients may be empirically determined, or for optimal control, analytically found by solving a complicated expression called the matrix Riccati Equation. The product of the matrix K and the conflict vector c is simply added to the estimate of $\hat{X}$ as an adjustment factor. I think you can see that if the actual sensory system characteristics of the body were altered (e.g. by labyrinthectomy or by wearing reversing prism glasses) or if the A matrix was suddenly changed in an unusual way (as when going into weightlessness; as shown in Figure 1, many of the terms in A are gravity dependent), then the conflict vector c will be unusually large, and the estimated state of the body $\hat{X}$ will not equal the actual state of the body X, until the appropriate term in the internal model has been changed by the CNS. Until then, the CNS would generate erroneous state estimates (disorientation illusions) and associated reflexes (m).

Presumably, the CNS is equipped with a means of identifying A,B, and S, and upadating the $\hat{A}$, $\hat{B}$, and $\hat{S}$ internal model matrices. The CNS could be doing this continuously, or might be "tipped off" by a persistent increase in the length of the conflict vector c. Of course, the only means the CNS has at its disposal for reidentification of the actual (altered) A, B, and S body characteristics is to observe motor outflow, and the resulting sensory input. The CNS cannot identify A, B, and S, and thereby update $\hat{A}$, $\hat{B}$, and $\hat{S}$, without active movement, unless external disturbances are present, and it is willing to <u>assume</u> something about the disturbance characteristics. But here, we are assuming that the externally applied forces η_e are <u>unpredictable</u>. So these sorts of arguments support Dick Held's notion that active body motion is a sufficient condition for adaptation to sensory rearrangement, and predict that the CNS should <u>not</u> be able to adapt to a white noise input motion, at least in terms of generating a correct body state estimate. See Figure 7.

I turned all this into a model for motion sickness by adopting the approach shown in Figure 8. The box marked "conflict tolerance criteria" $c^T c$ is just a mathematical way of measuring the length of the conflict vector in a mathematical "direction" to which the subject has demonstrated sensitivity. The output of this box is a scalar variable which serves as input to a (probably nonlinear) low pass filter, which accomplishes temporal averaging. The output of the averager, when added to any extrinsic factors influencing symptomatology level, produces symptoms when a threshold value is exceeded. All this just says that sensory conflict has to be high for some period of time before symptoms start to occur. Since each of the symptom classes in motion sickness appear to exhibit somewhat different dynamics, the blocks drawn apply only to nausea. Sweating and pallor, etc., are presumably triggered by the same conflict signal, but may be associated with different final (uncommon) pathways.

This sort of modelling exercise certainly suffers from being terribly abstract, I know. And the notation is obscure to those not trained in engineering. But it seems to me to be a start at a synthesis of some sort of general theory which takes off from an aspect of the human condition: the particular class of movement control task that the CNS is faced with. With respect to motion sickness, several general conclusions can be drawn:

(a) Perhaps there are fundamentally two types of motion sickness: that produced by changes in body or sensory system characteristics (i.e. sensory rearrangement, alterations in the A, B or S matrices); and also that produced by unpredictable externally imposed motion. The CNS appears to have some capability to reidentify new models to eliminate sickness and illusions in the former case. However, in the latter case, the conflict vector will remain high. Consequently, it makes sense that:

(b) Perhaps not all motion sickness adaptation takes place by reidentification of the internal models, particularly in cases where body characteristics change in an extreme way, or external motions are unpredictable. Sickness could, after all, also be reduced by increasing the CNS' tolerance to conflict (formally, a reduction in the eigenvalues of the T matrix in the model). A general model for motion sickness probably ought to allow for both possibilities.

(c) The important functional role of the "conflict" signal is likely to adjust our orientation perceptions and resulting motor outflow, and not to produce motion sickness. (Damn Triesman, anyway!) But the magnitude of the conflict signal would normally be small. I find the idea attractive that the uptake or deactivation mechanisms associated with conflict signal neurotransmitter systems are overwhelmed when conflict signals increase. Perhaps the averaging dynamics in the final "uncommon" pathways are associated with a diffusion process.

(d) It is interesting to note that the model formulation shows that motor outflow, m, should not always be directly correlated with the efferent copy signal $\hat{S}\hat{X}$, unless the desired orientation X_d is held constant. Put another way: if the desired body orientation is changing, the appropriate motor outflow depends on where you want the body to go, as well as to what you think it is doing at the moment, whereas the expected sensory signal (efferent copy) is only dependent on one's estimate of body state and not directly on commanded orientation. If one postulates that the CNS internal model is just a dictionary of matched pairs of traces of command signals and expected sensory inputs, then listed under each command signal must be traces of expected sensory inputs sub-classified according to the current estimated orientation of the body. This increases the size of the dictionary considerably. Isn't it more parsimonious to assume that the CNS merely retains the information necessary to generate the necessary paired traces, rather than the trace pairs themselves?

I suspect I've taxed your patience enough with this, so let me move on to more empirical topics. Although my Payload Specialist application wasn't successful, I've had the chance to accumulate several hours of time in weightlessness (albeit 30 seconds at a time) on NASA's KC-135 zero g airplane, as part of our Spacelab crew training and baseline testing program. I wish we'd all had the opportunity to experience this before we wrote our Academy report last year. One byproduct: remembering the suggestion you made at Snowmass, I made up a pair of vision restricting glasses by taping over all but the central 30 degrees field of view on a pair of sunglasses, and wore them on several occasions. Although I didn't experience any motion sickness symptoms, with or without the glasses (probably because I had medicated myself with scop/dex), it was clear that the reduced field of vision forced me to make a great many more head movements while working in zero-g than I otherwise would have. I came away feeling that vision restriction is probably not such a good idea for actively working payload crew. It is probably simpler just to limit your head movements and not allow yourself to move around the cabin much. Your learning curve in the first few hours in weightlessness is extremely steep; when you first start to maneuver around it is particularly bothersome that your body doesn't move in the manner expected when you push off. It's very difficult to create the pure translatory motions we make on earth. It's interesting that vision plays a vital role in determining the sense of down, but people seem to differ a good bit with respect to the strength of their sense of the 'down' direction in weightlessness, even with similar visual inputs. We now have some data on this for the individual Spacelab crewmembers. It was also intriguing to note that Owen Garriott, who spent 60 days on Skylab-3, immediately knew how to get around, even though he hadn't been weightless in five years. In the middle of the aircraft there was an open area where we all could practice bouncing around. With a half dozen of us in there, each trying to do loops and spins, it was like a three dimensional football game with lots of body contact. But from the start, Owen was moving around smoothly and quickly, never colliding with anyone, and could execute impressive maneuvers just by managing his moment of inertia, something none of the rest of us could do without considerable practice.

On a slightly different subject, I've been giving a good deal of thought lately to the business of subjective scaling of motion sickness symptomatology, because of my interest in modelling the dynamics of motion sickness, and could use some good advice. One of our postdocs, Otmar Bock, and I, have been trying out a preliminary series of experiments using left-right reversing prism goggles and head movements as stimulus, and are attempting to study the extent to which subjects can be trained to consistently report their nausea and subjective well-being. We're keeping track of the head movement stimulus and would like to establish a modified ratio or interval scale à la Smitty Stevens. I know you worked on this sort of thing a few years ago, and was curious whether you've kept at it. I am frankly surprised how consistent our subjects seem to be. They report little trouble saying when they feel 'twice as good' or 'twice as bad' as some standard they adopt. Determining the scaling law for nausea is certainly a prerequisite for quantitative motion sickness measurement and modelling.

Needless to say, I'm anxious to run my theoretical notions past you again in more detail, and to hear what you've been up to in the last year or so. It turns out that I must travel to Edinburgh this fall for a sailing championship (September 3-7). If your invitation for a visit is still open, and your schedule permits, I may find a way to get to Manchester for a day or two either before or

(preferably) after the event. There is a chance I can finagle funds for part of the trip from one of our grants, but if not, I'll come up with some other way of covering it, if only from out of pocket. Transatlantic fare structures being what they are, though, I can save a good bit of money if I can pin down my travel dates soon.

I understand there has been a considerable discussion of seasickness lately in your magazine YACHTING MONTHLY, wherein two clinicians from Basingstoke make a pitch for the effectiveness of the drug Stugeron (cinnarizine). Attached is my own contribution to the confusion which appeared in SAIL last November. You might be amused that my article elicited more than a few letters and phone calls from individuals tormented by mal de mer who wanted to know why their physicians hadn't bothered to tell them about the conflict theory. When they ask for the details of the theory, of course, I tell them that they should go out and buy the readable, comprehensive, up-to-date reference on motion sickness: Reason and Brand (even though it costs more than a whole case of Dramamine). The Spacelab crew has done this. So if your Academic Press royalties from North America are significantly up this year, you probably owe me a dinner.

With very best wishes.

Very truly yours,

Chuck

Charles M. Oman
Helmholtz Associate Professor

Enc. Motion sickness model notes
Sail article

Sensory/Motor Conflict Model
for Motion Sickness

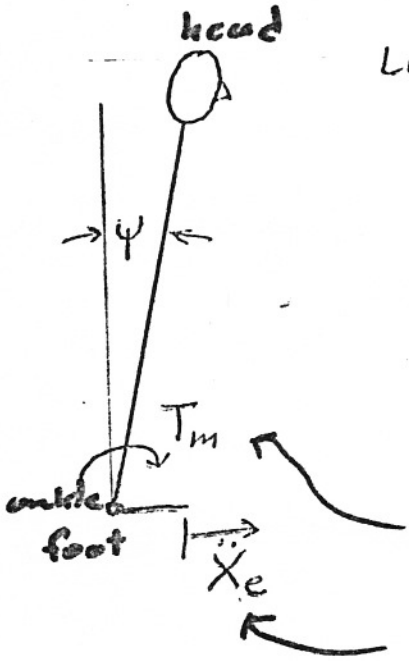
Viewgraphs

C.M. Oman
MIT 37-219

Presented at NASA/JSC Vestibular/Motion Sickness Workshop, Nov. '78

Figure 1

Example: Stabilization of Body Sway



Linearized Model:

$$\ddot{\psi} = -\frac{B_b}{I_e}\dot{\psi} + \frac{g}{h_e}\psi + \frac{T_m}{I_e} - \frac{\ddot{X}_e}{h_e}$$

where

$$I_e = I_{cg} + Mh^2$$

$$h_e = \frac{I_{cg} + Mh^2}{Mh}$$

Semicircular Canals (Conventional Steinhausen Model)

$$\ddot{\xi} = -\frac{\Pi}{\Theta}\dot{\xi} - \frac{\Delta}{\Theta}\xi + \frac{\ddot{\psi}}{D_1}$$

Utricular Otolith (Conventional model for otolith mechanics + neural adaptation)

$$\ddot{\delta} = -\frac{B_o}{m_o}\dot{\delta} - \frac{K_o}{m_o}\delta + \frac{(\rho_o - \rho_e)}{\rho_o}\left[\ddot{\psi}_d + \ddot{X}_e\right]$$

$$\dot{r} = \dot{\delta} + \frac{(1-K_a)}{T_a}\delta - \frac{1}{T_a}r$$

Figure 2

State Differential Equation.

for body sway dynamics

$$X = (\psi, \dot{\psi}, \xi, \dot{\xi}, \delta, \dot{\delta}, r) \qquad u = (T_m, \ddot{x}_e)$$

Body State Vector — Forcing Vector

Rewriting Equations in Figure 1 in matrix notation:

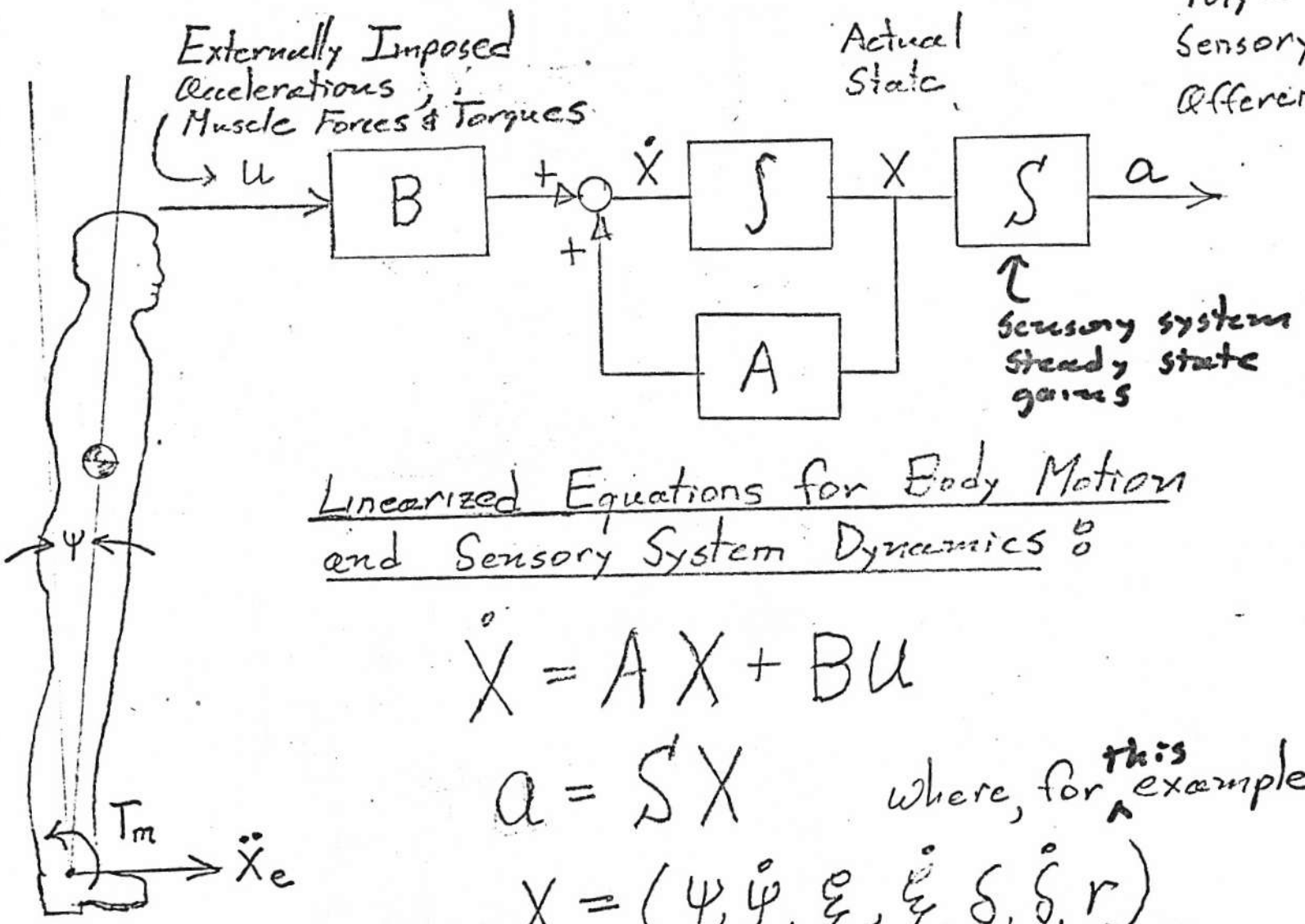
$$\dot{X} = \begin{bmatrix} 0 & 1 & 0 & 0 & 0 & 0 & 0 \\ -\frac{B_b}{I_e} & \frac{g}{h_e} & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 1 & 0 & 0 & 0 \\ -\frac{B_b}{I_e D_1} & \frac{g}{h_e D_1} & -\frac{\Delta}{\Theta} & -\frac{\pi}{\Theta} & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 1 & 0 & 0 \\ \left(\frac{d B_b (\rho_o - \rho_e)}{I_e \rho_o}\right)\left(\frac{d g (\rho_o - \rho_e)}{h_e \rho_o}\right) & & 0 & 0 & -\frac{B_o}{M_o} & -\frac{K_o}{M_o} & 0 \\ 0 & 0 & 0 & 0 & \frac{1-K_a}{\tau_a} & 1 & -\frac{1}{\tau_a} \end{bmatrix} X + \begin{bmatrix} 0 & 0 \\ \frac{1}{I_e} & -\frac{1}{h_e} \\ 0 & 0 \\ \frac{1}{I_e D_1} & -\frac{1}{h_e D_1} \\ 0 & 0 \\ \left[\frac{d T_m (\rho_o - \rho_e)}{I_e \rho_o}\right] & \left[\frac{\rho_o - \rho_e}{\rho_o}\right]\left(1 - \frac{d}{h_e}\right) \\ 0 & 0 \end{bmatrix} u$$

It looks hairy, but note the form is simple:

$$\dot{X} = A X + B u$$

where A and B are the two big matrices above.....

Figure 3



Linearized Equations for Body Motion and Sensory System Dynamics

$$\dot{X} = AX + BU$$

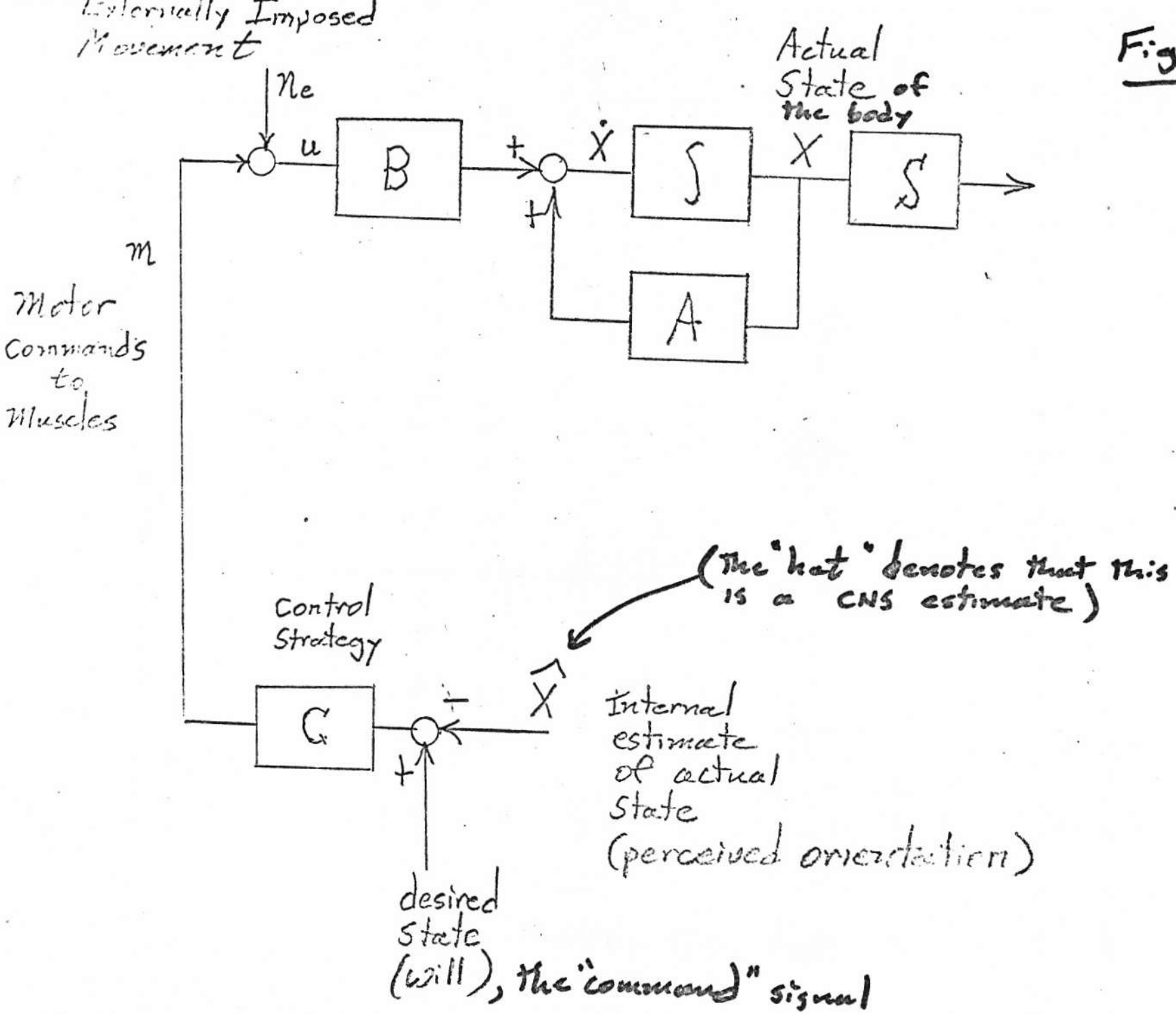
$$a = SX$$

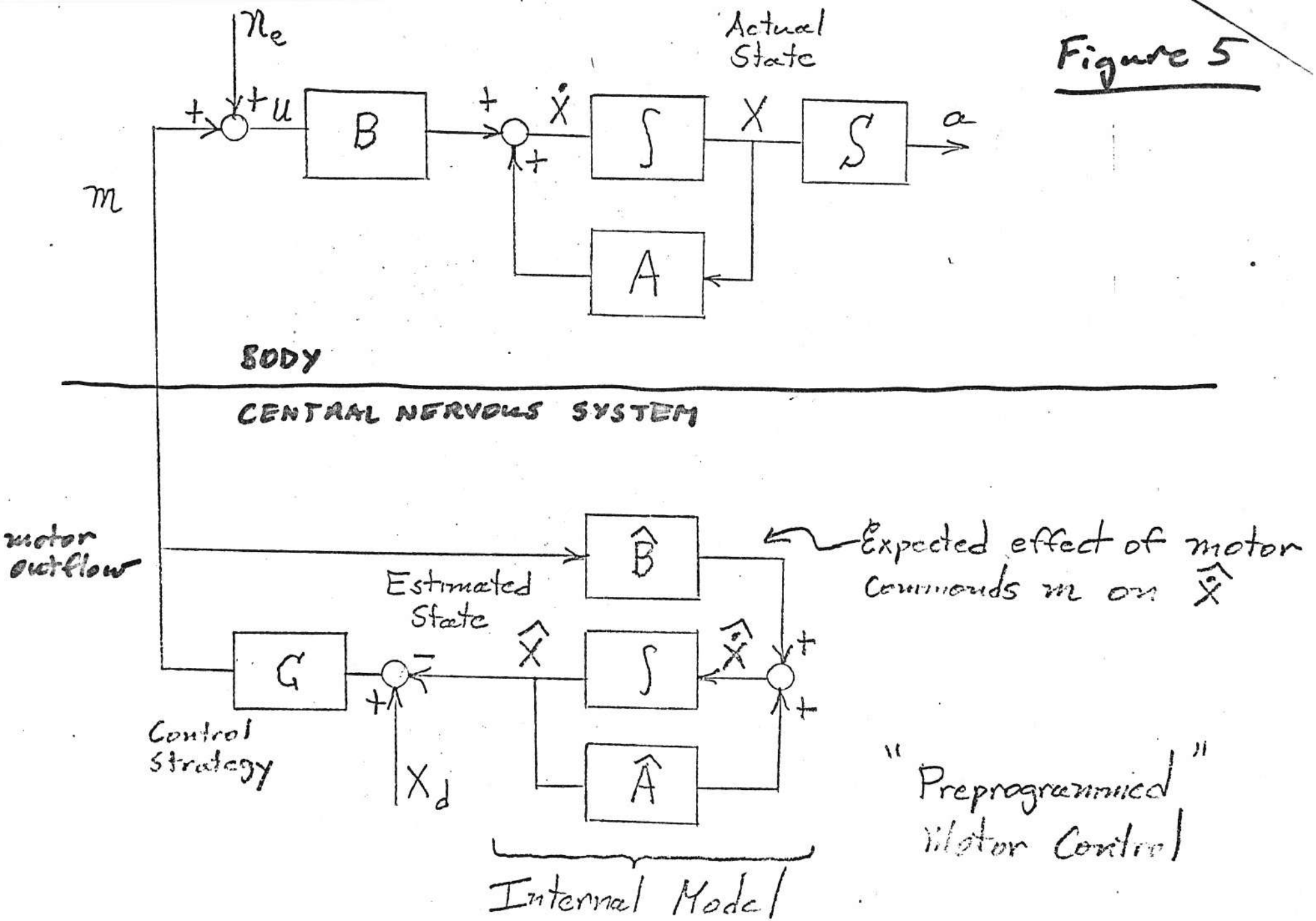
where, for this example

$$X = (\Psi, \dot{\Psi}, \xi, \dot{\xi}, \delta, \dot{\delta}, r)$$

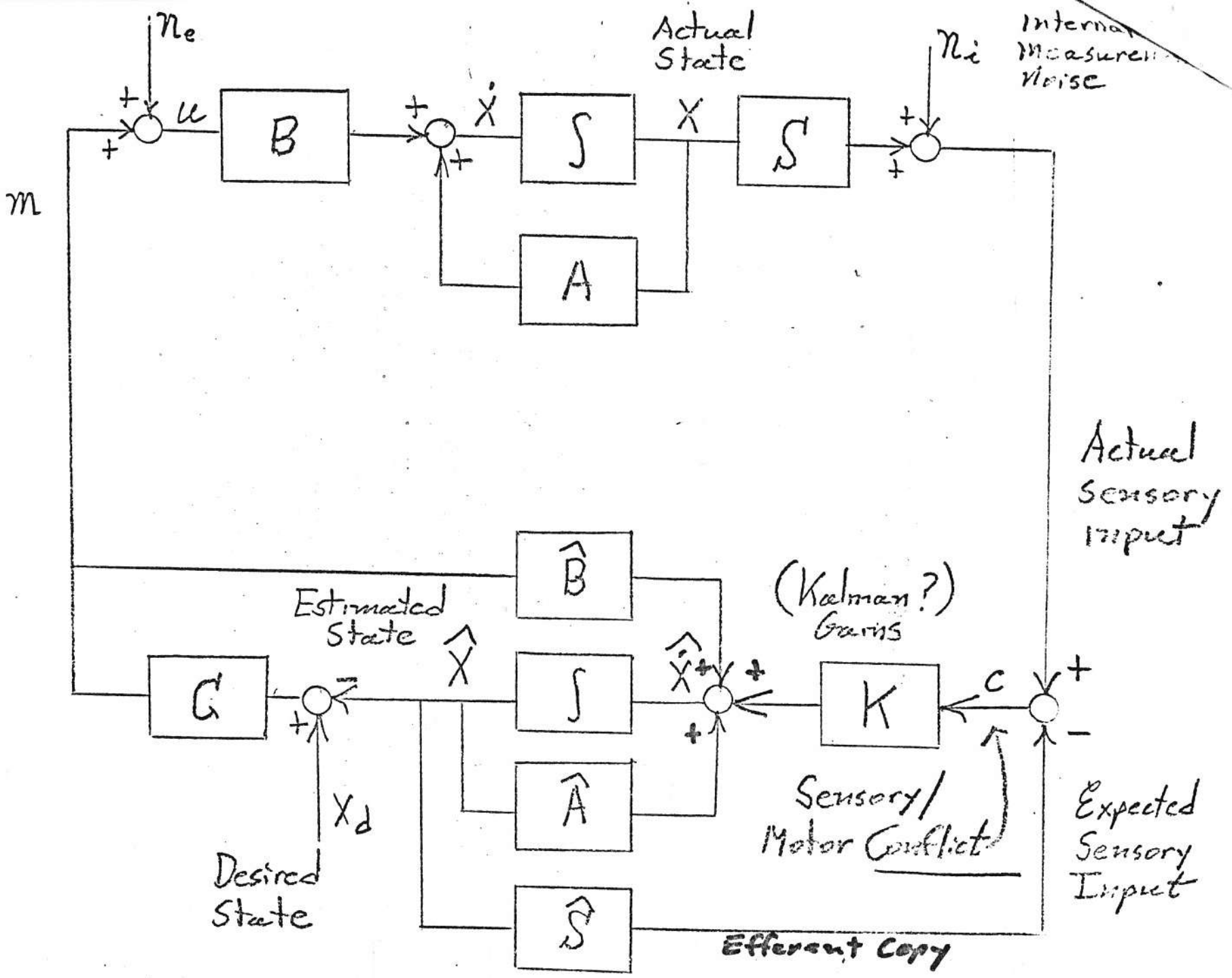
$$u = (T_m, \ddot{X}_e)$$

Figure 4


Externally Imposed Movement
n_e
u
B
+
+
$\dot{X}$
$\int$
X
$\int$
Actual State of the body
A
m
Motor Commands to Muscles
Control Strategy
G
−
+
$\hat{X}$
(The "hat" denotes that this is a CNS estimate)
Internal estimate of actual state (perceived orientation)
desired state (will), the "command" signal



If $n_e = 0$ and $\hat{A} = A$, $\hat{B} = B$, then $\hat{X} = X$ and good control of the body is achieved open loop. But these assumptions are unrealistic



Output Feedback (Optimal) Control System

Figure 6

Figure 7

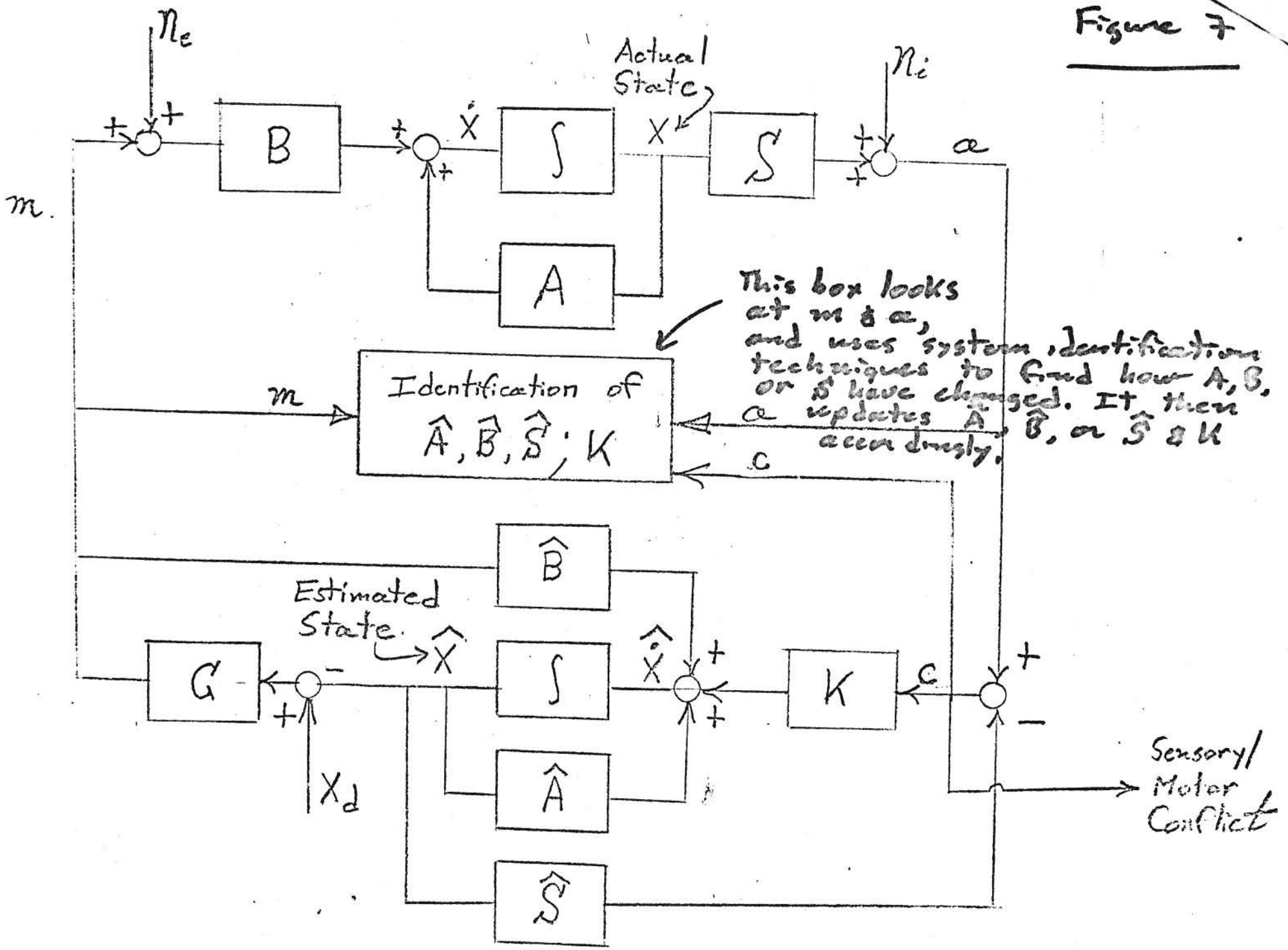
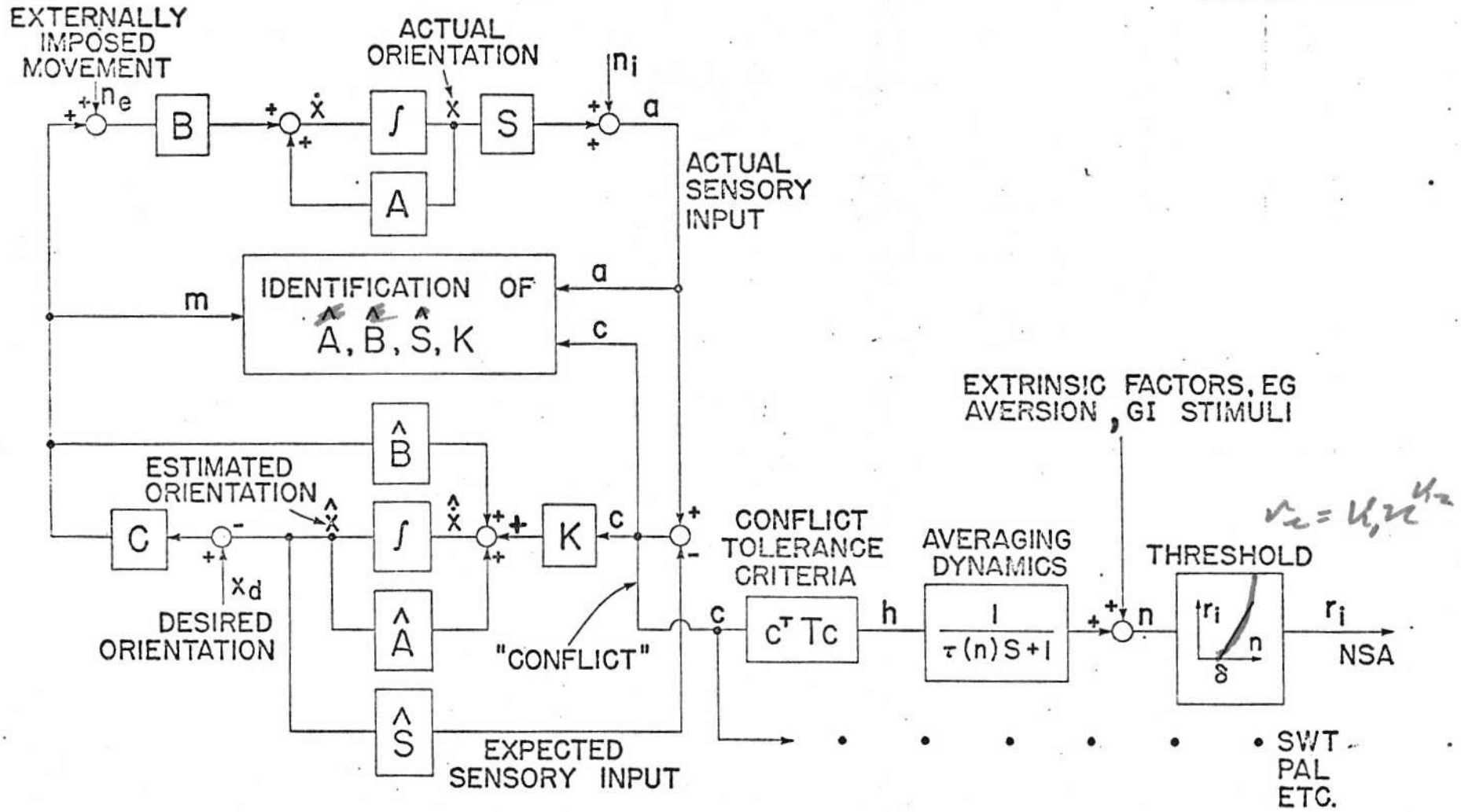
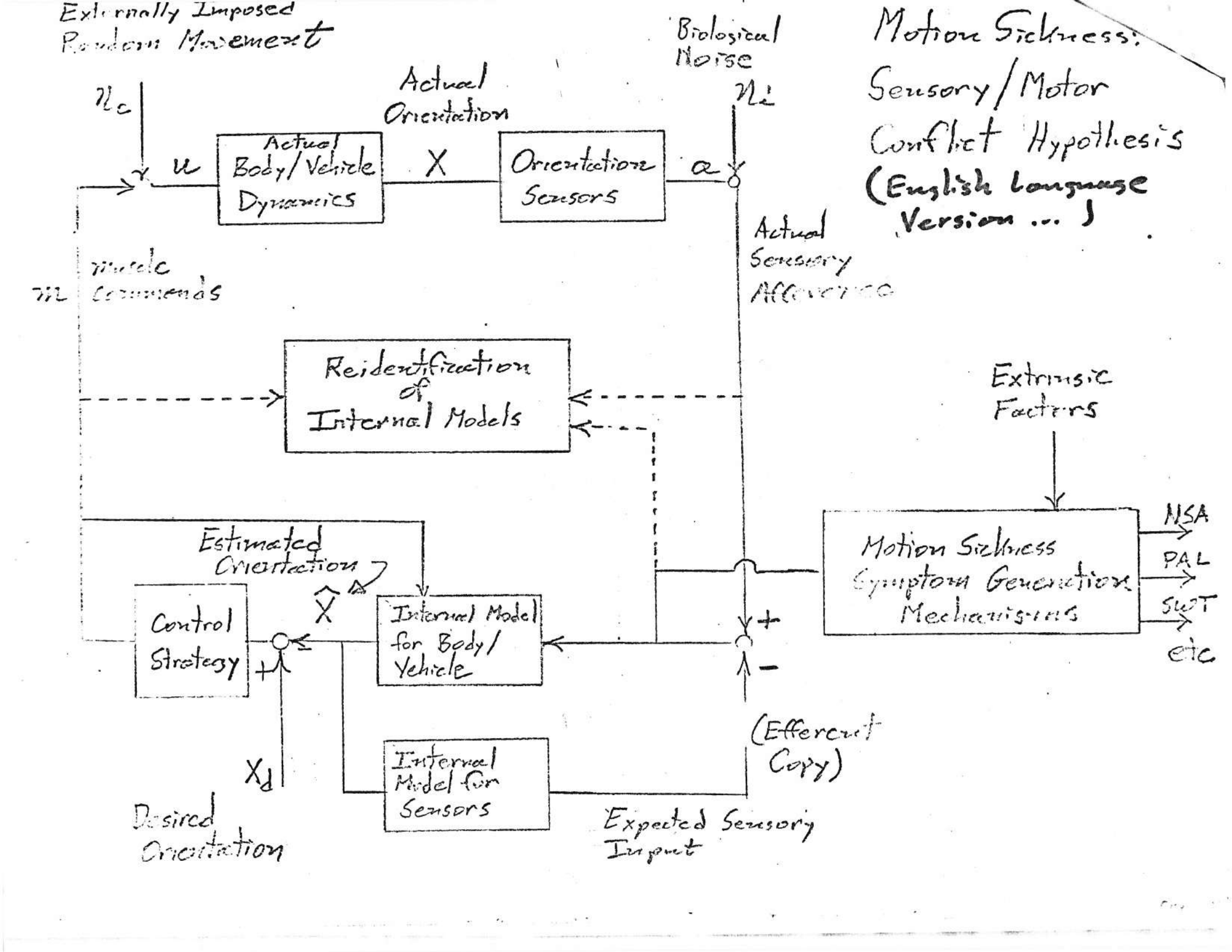

n_e
Actual State
n_i
B
$\dot{X}$
$\int$
X
S
a
m
A
This box looks at m & a, and uses system identification techniques to find how A, B, or S have changed. It then updates $\hat{A}$, $\hat{B}$, or $\hat{S}$ & K accordingly.
m
Identification of $\hat{A}, \hat{B}, \hat{S}$; K
a
c
$\hat{B}$
Estimated State
$\hat{X}$
G
$\hat{X}$
$\int$
$\hat{X}$
K
c
X_d
$\hat{A}$
$\hat{S}$
Sensory/ Motor Conflict

Figure 8


EXTERNALLY IMPOSED MOVEMENT
n_e
B
$\dot{x}$
$\int$
$\hat{x}$
S
n_i
a
ACTUAL ORIENTATION
A
ACTUAL SENSORY INPUT
m
IDENTIFICATION OF $\hat{A}, \hat{B}, \hat{S}, K$
a
c
$\hat{B}$
ESTIMATED ORIENTATION
C
$\hat{x}$
K
c
x_d
DESIRED ORIENTATION
$\hat{A}$
"CONFLICT"
$\hat{S}$
EXPECTED SENSORY INPUT
CONFLICT TOLERANCE CRITERIA
$c^T Tc$
h
AVERAGING DYNAMICS
$\frac{1}{\tau(n)S+1}$
EXTRINSIC FACTORS, EG AVERSION, GI STIMULI
n
THRESHOLD
r_i
δ
r_i
NSA
SWT
PAL
ETC.


Motion Sickness: Sensory/Motor Conflict Hypothesis (English Language Version ...)
Externally Imposed Random Movement
Biological Noise
n_c
n_i
Actual Orientation
u
Actual Body/Vehicle Dynamics
X
Orientation Sensors
a
Actual Sensory Afference
n_c
muscle commands
Reidentification of Internal Models
Extrinsic Factors
Motion Sickness Symptom Generation Mechanisms
NSA
PAL
SWT
etc
Estimated Orientation
$\hat{X}$
Control Strategy
Internal Model for Body/Vehicle
+
−
(Efference Copy)
X_d
Desired Orientation
Internal Model for Sensors
Expected Sensory Input

SENSORY/MOTOR CONFLICT MODEL

Normal Active Body Movement Control

$(\hat{A}, \hat{B}, \hat{S}) = (A, B, S)$

X_D, X can be large (e.g. dancing, acrobatics)

C small

Motion sickness = 0

SENSORY/MOTOR CONFLICT MODEL

RESPONSES TO PASSIVE MOTION AND SENSORY REARRANGEMENT

I. SOME REDUCTION IN T TOLERANCE MATRIX

ALSO: S (EFFERENTS) ?
K

COST: REDUCED CONTROL SYSTEM PERFORMANCE

RESULT: SOME GENERALIZED REDUCTION IN SICKNESS SUSCEPTIBILITY

SENSORY/MOTOR CONFLICT MODEL

RESPONSES TO PASSIVE MOTION AND SENSORY REARRANGEMENT

II. SENSORY REARRANGEMENT $(A, B, S) = (A', B', S')$

DURING ACTIVE MOVEMENT CONTROL

$$(\hat{A}, \hat{B}, \hat{S}, K) \Rightarrow (\hat{A}', \hat{B}', \hat{S}', K')$$

EXAMPLES: PRISM ADAPTATION

SPACE SICKNESS

TIME REQUIRED BEFORE MOTION SICKNESS = 0 : DAYS

BUT:

POSSIBLE RETENTION OF SETS OF LEARNED MODELS

$$(\hat{A}', \hat{B}', \hat{S}', K')$$

AND USE IN APPROPRIATE CONTEXT

EXAMPLES: EXPERIENCED DRIVERS, PILOTS

SPECTACLE WEARERS

TIME REQUIRED: SECONDS

SENSORY/MOTOR CONFLICT MODEL

RESPONSES TO PASSIVE MOTION AND SENSORY REARRANGEMENT

III. PASSIVE BODY MOVEMENT (η_E)

η_E RANDOM: A, B UNALTERED

C POTENTIALLY LARGE

MOTION SICKNESS $\neq 0$

η_E PREDICTABLE

A, B AUGMENTED TO INCLUDE OSCILLATOR DYNAMICS (GROEN PATTERN CENTER)

EG. "SEA LEGS"