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"Characterization and Etiology of Space Motion Sickness"

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CHARACTERIZATION & ETIOLOGY OF SPACE MOTION SICKNESS

Whenever man is placed in environments of unaccustomed motion, real or simulated, a sizable percentage of the population will develop the characteristic syndrome of motion sickness.(1) This is at least a nuisance to many individuals and a significant problem to modern military forces, hence much of the study of the problem has been sponsored by the military, especially in World War I and II. (2) Dr. Graybiels' work in the U.S. Navy is an archtype of such research. With development of numerical scoring of signs and symptoms and means of rapidly inducing motion sickness, such as coriolis effects, motion sickness research ~~became~~ has become almost stylized. It was possible to develop such scoring only because susceptible individuals develop characteristic signs and symptoms on continued exposure to an environment which produces major sensory conflict.

Fig. 1 Graybiel Rating

If exposure is continued, vomiting and retching may be prolonged, sometimes with prostration. After varying amounts of exposure, the majority of subjects will develop resistance to the specific stimuli. Several medications, habituation and training, are effective in prevention or treatment to varying degrees. In addition to the above symptoms Graybiel proposed a 'sopite' syndrome which may be intertwined with motion sickness or occur under prolonged mild stimuli or as a variant of motion sickness under strong stimuli. Features of this syndrome, as proposed, include yawning, drowsiness, disinclination for physical or mental work, and lack of participation in group activities.[3]

Prior to spaceflight it was predicted that a conflict between the gravity sensitive statolith organs and independent canals would occur in the weightlessness of spaceflight and produce a variety of symptoms. When the second, and several subsequent cosmonauts complained of disorientation, illusions, malaise, nausea and vomiting[4] and this was followed by similar complaints later in the Apollo Program, it was not unreasonable to consider this

Motion Sickness and treat it accordingly. However, by the end of Skylab there was reason to doubt that the sickness in space was absolutely identical to that on earth since there was little correlation between susceptibility on earth and in space, the medications effective on earth were at best questionable in space, and after a few days in space individuals became remarkably resistant to coriolis stimulation, a non-specific adaptation.(5) After the third flight it was obvious that the Shuttle Program would also have to contend with this problem which, under pressure of media and investigators, became 'serious, dangerous, threatening, etc.' There were repeated attempts to document signs and symptoms by means of questionaires and debriefings but the results left much to be desired.

Operational Inflight Investigation:

Prior to STS-4 a few people in the J.S.C. Astronaut Office and in Flight Medicine felt that objective inflight investigation of the problem with major emphasis on operational concerns was necessary, especially with accumulating anecdotal information

describing further differences between sickness inflight and on earth.

An operationally oriented program was mounted on Shuttle flights STS-4 thru STS-8, with physicians on STS-7 & STS-8 to perform the studies. Some studies of this series have been continued to the present. This investigation used accepted objective clinical procedures where possible and had the major goals of:

1. Clinical characterization.
2. Investigation of etiology.
3. Investigation of possible treatment.

A listing of studies is given in **Table II**. The following is largely based on this investigation which includes the author's personal observation and study of six episodes of space motion sickness on two flights and, in some areas, on additional anecdotal information from Astronaut flight experience. This does not include anecdotal or other information from SL-1 or D-1 missions which will be presented by their investigators.

Symptoms:

Motion Sensitivity:

There is an amazingly wide and variable range of symptoms in S.M.S. but typically, the first indication is hypersensitivity to angular head motion, either head alone, or from combined head and body motion. In many subjects this sensitivity is predominantly in the pitch plane, in others it is in yaw, but in every case is also present in all other angular axes. This hypersensitivity may become noticeable from zero to 1-2 hours after exposure and can only be described as a thoroughly unpleasant sensation not to be repeated if possible. It does not produce visual disturbance or illusion nor does it obviously produce stomach symptoms as, for example, does out of plane head motion in a spinning chair. If anything, it is increased with eyes closed. The sensation strength is directly related to the velocity of movement.

Translation, even reciprocating translation, does not produce these symptoms. This hypersensitivity typically increases to a plateau in several hours and remains at that level until

resolution when it rapidly diminishes. One simply wants a quiet immobile spot. There is frequent somnolence which increases during this time, as well as lethargy and loss of initiative.

Illusions, Visual Disturbance, Orientation:

This is reported as a major symptom in the Russian Program ( ) and in some of the Apollo experiences and included illusion of both position and movement. With the exception of SL-1 and D-1 crews I have questioned all Shuttle Astronauts after flight and have been unable to elicit any admission of either visual disturbance or illusion on launch or orbit except in one pilot who was not motion sick and claimed an illusion of being in a static pitched down position.

Much has been made of the 'egocentric' ability or referencing surroundings to one's own axis, for example, the ability to place the earth above one's head rather than being inverted above the earth. I was able to do this easily but it did not prevent S.M.S. in myself nor in several others questioned. Sensitivity to scenes out of alignment with one's own reference, e.g., inverted

earth, inverted crewmen, etc., appears to have been disturbing to a few but not the majority. A common illusion unrelated to S.M.S. may occur in aircraft pilots observing the earth while strapped in the CDR or PLT seats with the Shuttle nose down. One feels as if it is pitching further down. This may be avoided by releasing the seat belt. Dr. Lackner has reported similar experience in Zero-g aircraft.

Gastro Intestinal:

Signs and symptoms may also appear from minutes to several hours after weightlessness and often consist of a very brief bout of unproductive retching but usually of sudden vomiting without nausea or other prodrome. The vomiting is strenuous, brief, and appears to empty the stomach of whatever contents are present, undigested but rarely they are bile stained. Subjective relief is commonly claimed after vomiting. In the absence of eating or drinking these events may be repeated from one to several times, usually with hours of spacing between events with clear vomitus. Vomiting is not prolonged, there are no dry heaves nor frequent

bouts. Typically all significant amounts of ingested food or drink will be vomited, usually within thirty minutes to an hour or more. The majority of subjects deny nausea, but in some this is a major symptom or may be a presenting symptom. This nausea may wax or wane but in my experience is not related to other activity although some motions will be avoided. Anorexia is almost universal. A variety of non-specific epigastric symptoms have been recorded, the most common being a 'knot in the stomach'. Lower bowel functions as judged by flatus and defecation seem normal.

There have been attempts to link the vomiting to either visual or motion inputs. Such inputs may trigger an impending event, but ingestion of food seems to be the only reliable cause for vomiting.

Autonomic Signs:

There is virtually no incidence of sweating, and flushing is more common than pallor. A current study to document incidence and extent of pallor/flushing is underway using chromatically calibrated and analyzed color photographs.

Other:

Headache is common, usually mild, non-specific and with a variety of locations. Malaise typically increases the first few hours and plateaus ~~while~~ there is a marked loss of initiative. Somnolence is very common and may be severe with individuals sleeping deeply at the slightest opportunity. This is frequently a symptom which develops early and persists.

Effects of Activities:

The stories are endless in this area so only the most consistent are offered. Demanding activities such as the Commander's duties, responsibility for satellite launch or RMS operations, appear to reduce the perceived discomfort if not actual level of S.M.S. Another common bit of lore is that excessive movement early on orbit may precipitate or increase the symptoms. In any event, cessation of activity, even sleeping, may decrease the discomfort but does not cure the disease.

Objective Studies:

Because of the unique relation between eye motion and the greater vestibular system, electro-ogulography was most intensively studied of all investigations.

Sick/Non-Sick Incidence:

Two interrelated questions are the incidence of S.M.S. and the horizontal overlap of symptoms in those affected and not affected and the presence of symptoms from other causes. Incidence depends upon point of view and has been placed very high by investigators and others seeking funds or other resources, typically 50% or more, and much lower by those directly involved, say 30%. My own estimate is 40% or less.

I was much more impressed by the obvious difference between those with and without S.M.S. in contrast to the gradation of symptoms I had predicted. Also there is ample stimulus available for ordinary motions sickness, e.g., vertical launch and visually inverted flight with up to 3.5g eyeballs down terminating in weightlessness. It didn't surprise me that people came off the

flight deck with their breakfast in the sick sack. There are also a host of other new sensations so diagnosis of S.M.S. must be made with some care, especially from the ground.

Horizontal and vertical EOG were recorded during launch and entry on one flight and during entry on two flights with 3 subjects. Horizontal EOG and head motion were monitored during 3 ascents and entries with a total of 4 subjects.

Conventional calibration, electrode configurations and equipment standards were employed. Standardized voluntary head oscillations with eyes open and fixed on a target, and with vision occluded, were made before, during, and after ascent and entry, as well as continuous recordings during launch and entry. No abnormalities were seen, not even an occasional beat of nystagmus.

On orbit a more or less conventional EOG exam was performed, without Hallpike maneuver and caloric stimulation, and with voluntary head **Fig. 4** oscillation substituted for the oscillating chair. **Fig. 5**.

Again conventional standards were adhered to although the equipment had to be designed to fit the situation. Some - records were made on orbit with - pre- and post-flight controls. This series can be summarized by one word - normal, with exception of two isolated records containing distortion during the head oscillation which seemed most likely to be artifactual.

Another major effort was made to document autonomic changes during Space Motion Sickness including facial color, pupillary size, temperature, heart rate and blood pressure. These have proven extraordinarily difficult to obtain for non-technical reasons and we do not have an adequate statistical sample to date, however attempts continue. As noted, observation shows pallor or flushing depending upon the individual, with apparently normal pupillary size. Ambulatory heart rate and blood pressure from one subject showed them to be remarkably.low as the symptoms plateaued the first day.

Gastric and intestinal motility studies, while difficult, have been the most productive of all. I shall discuss these in some

detail in a second presentation, but they will be summarized here. Dr. Thagard, as part of our studies on STS-7, observed that bowel sounds were absent during the course of S.M.S. and this finding has been subsequently confirmed by auscultation in every case observed, as well as all those objectively studied.

Studies consist of recording sounds from right and left upper quadrant preflight **Fig. 7** during and after SMS inflight in parallel with unaffected controls. The records are semiquantitatively scored by counting the rate of audible events by standard criteria. Weightlessness does not greatly alter the rate or quality of bowel sounds in those unaffected by S.M.S. although some individuals may be hyperactive the first day. Conversely S.M.S. greatly reduces or in some cases eliminates all detectable activity during the course of the syndrome. **Fig. 8.** On recovery, activity is normal or sometimes hyperactive. There is some evidence for rebound activity for the first hours after recovery followed by normal activity. To date, this phenomenon is the only

one which bears a constant relation to the presence of this syndrome.

Performance During S.M.S.:

This, as always, is the most difficult evaluation to make. Even under normal circumstances, tests of performance are, at best, tenuously related to actuality. While it is obvious that a person is hors de combat during vomiting, this is brief. Conversely, trained astronauts have in every case, except a scheduled EVA on Apollo, performed assigned tasks for which they were trained. It isn't necessarily easy, nor do people affected seek extra duties during the period, and many of the tasks require concentration as well as good neuro-muscular and eye-hand coordination. There have been cases of payload specialists, who have not had the rigors of astronaut selection and training, being unable to complete all assigned tasks.

In an effort to study effects of S.M.S. on performance two areas have been examined -- neuro-muscular performance and mental processing. The first consisted of returning to a fixed linear position of hand or arm **Fig. 9.** after voluntary displacement from

it, and of manual tracking of a visual target on a linear scale which moved in a series of regular and aperiodic functions. A second study uses the relatively common Sternberg test **Fig. 10.** which consists of the timed indication of presence or absence of a single digit in a previously displayed number. Neither of these tests have shown any decrement in performance in the few cases we have been able to examine to date.

Temporal Profile of Symptoms:

As noted, with an exception which will be treated later, onset of symptom occurs within minutes to 1-2 hours of exposure to weightless-ness. This progresses in intensity over a period of hours to a plateau which for a given condition remains stable. There are typically both head and gut symptoms although one or the other may predominate and, in some subjects, the gut symptoms may be the only ones recognized. In every case the gut remains quiet. Vomiting is often more frequent at the beginning and after one or two episodes may not recur in the absence of intake.

The resolution of symptoms is typically sudden and dramatic and most frequently occurs between 30-48 hours, but has been as short as 12 hours, and possibly as long as 72 hours. During and after resolution there is usually a marked change in attitude, loss of malaise, return of stomach activity and usually appetite, and marked decrease in motion sensitivity usually in a matter of hours or less. There may be some residual motion sensitivity which decreases to normal over the next 2-3 days. With determined effort this sensitivity can be aggravated. Anorexia also may remain, but hunger is more common. At this time or in the following days resistance to all forms of motion sickness develops including the out of plane head motions in the rotating chair as was first demonstrated on Skylab.

Delayed Onset:

There was a sub-group of 4 crewmen to my knowledge who had significantly delayed onset of symptons, one for 48+hours. I observed this crewman and he was very active and sympton-free for the first 2 days, yet developed a moderate case which persisted for

24+hours. The things in common in these four was medication with ScopDex, with onset of symptoms after discontinuation of the medication. This is the most convincing evidence I have seen for efficacy of any drug but this represents only a small number of the subjects taking this medication. It is probably significant that symptoms were not prevented, only delayed.

Reentry and Post-Flight:

There has been no recurrence of symptoms after landing in the American program, although this is reported to be common in the Russian Program. **(12A)**. During reentry and for hours thereafter head turns may provoke a sense of disequilibrium in some subjects, including those not affected by S.M.S., but never with the sense of unpleasantness experienced by those with S.M.S. inflight. One subject however claims to have developed motion sickness on reentry while doing vigorous head motions as part of an investigation. Some subjects have noted an illusion of translation on head turns after re-exposure to 1g and this has caught the attention of some investigators. I could not elicit the same

phenomena inflight from any subject including one excellent observer who experienced it briefly on return.

While several changes in sensation occur postflight, none can in any way be characterized as sickness. Most striking of these postflight changes, which has not been properly studied, is resistance to all forms of motion sickness or even disequilibrium. There have been anecdotes of such increased postflight resistance to motion sickness, including those without motion sickness, especially in aircraft. I can vouch that this is correct for 19-days after my first flight I tried every maneuver possible in the T-38, repetitively, and could elicit nothing. Two of us also rode the chair with head motions postflight, without any effect, although on the day of landing I had been hypersensitive to it. This lack of sensitivity appeared to last for weeks but is one of many questions which need answers.

The question of acquired resistance to S.M.S. has not been adequately documented. At one time it was considered part of flight readiness to have gone through informal but vigorous

acclimatization by violent repetitive maneuvers in the T-38 and in some cases prolonged sessions in the spinning chair. Some of the subjects most resistant to motion sickness suffered most from S.M.S. Conversely there is increasing evidence, largely undocumented, that prior spaceflight produces resistance to S.M.S. This is supported by several motility studies I have done in reflown individuals.

Previous flight appears to have no effect after a period of 10 years or more. For those who have flown within 2-3 years there is wide individual variation with some showing relatively small efffects even with flights as recent as 7-months, while another was symptom free on his second flight after a delay of 2+years. In some there is no reduction in symptoms but a reduction in duration while in others there is a significant reduction in severity of symptoms. In no case has there been a failure to adapt to weightlessness nor has there been development of symptoms.

Etiology:

There is not time to review theories of etiology of motion sickness on earth except to say that sensory conflict currently best fits our existing knowledge. We can find a number of situations involving major sensory modalities: vision, somatosensory, vestibular which in continued conflict will produce the classic progressive symptom complex of motion sickness. How the conflicting temporal profiles of neurological impulses is translated into the symptoms remains unknown.

We have also seen that there are significant differences between S.M.S. symptoms and the classic motion sickness symptomatology. There are other factors to consider in S.M.S. such as the large and rapid cephalad fluid shifts on exposure to weightlessness. Taking the symptoms of S.M.S., malaise, lethargy, headache, sudden vomiting and reports of illusions, one could not reasonably exclude the possibility of malfunctioning end organs nor even of increased intracranial pressure. At the time the inflight investigation was started and without the previously described data

and experience, the following possible causes of S.M.S. had to be considered and investigated. **Fig. 26A.**

Disorded Function	Anomalous Signals
Vestibular Hydrops	Visual
Increased I.C. Pressure	Vestibular S.C. Canals
Cervical Vertigo	Statolity Organs
	Somatosensory
	Visceral

They were based on clinical experience and a word of explanation may be in order for each.

Vestibular hydrops or in this case pseudo Meniere's disease could result from a sudden shift of labyrinthine fluid pressure or composition. In the same way changes in I.C.P. or composition were also possible. **(27A)** **(27B Facies)** It was known that in the absence of hydrostatic pressures several liters of fluid are shifted from legs alone and that part of it was retained as edema

in facial tissue and in mucous membranes. Similar internal changes could not be ruled out.

Cervical vertigo is a variously described but apparently real syndrome usually resulting from trauma to the neck's somatic sensors and may produce vertigo, nausea and other motion sickness symptoms. It is known that significant expansion of the intervertebral discs occurs in weightlessness, usually beyond that seen in bed rest on earth. There is also a change in the carrying angle of the head in weightlessness. These two factors could conceivably produce distortion in cervical sensors and their signals.

Weightlessness can also produce anomalous signals in our normal earth based sensory systems. There was little reason to think that weightlessness would directly affect the visual system - which in many ways remains the standard of comparison. Conversely many correct scenes are inconsistent with previous experience and might well produce symptoms, e.g., rapid angular maneuvers or positions incongruent with local orientation which are not possible on earth.

There is an inherent conflict between canal and statolith organs in weightlessness for the dynamic angular responses of each overlap and weightlessness will grossly distort the statolith organ's signal. While the static component of this signal is correct it will conflict with previous experience and with visual and possibly other sensory signals.

Many somatosensory signals will have never been experienced before. Relatively little is known of visceral signals beyond the fact that they occasionally reach consciousness during motion, particularly vertical accelerations, and that they are capable of producing a variety of upsets.

An investigational program was designed to study as many potential etiologies as possible with minimum resources. For example, EOG may provide information on several of the above categories. Because of its nature, determination of etiology was not possible with techniques currently available to us, rather it was possible to reasonably exclude most of the possibilities and focus on

the most probable cause. There is not time to give the usual details of procedures or detailed results so only summaries are offered.

Treating the potential causes as listed:

Vestibular Hydrops:

Illusions and visual field disturbances denied, clinical neurological exam normal, EOG exam normal, no difference in audio threshold sensitivity or audio evoked potentials between those affected and unaffected, and no difference in fluid shifted from legs in those with and without S.M.S.

Increased CNS Pressure:

Illusions and other neurological disturbances denied, clinical neurological exam normal, no changes in fundus, EOG normal, one intra ocular pressure normal, audio evoked potentials including mid-latency studies were normal, eye hand tracking was normal, one visual evoked potential normal, no difference in fluid shifted from legs in those with and without S.M.S.

Cervical Vertigo:

Illusions and other neurological disturbances denied, clinical neurological exam normal, EOG normal, no difference in height increases in those with and without S.M.S., cervical loading without affect in one subject.

In summary, there was no positive evidence for altered sensory or CNS functions. When the potential role of various sensory inputs are examined there is less hard evidence and subjective symptoms are open to many interpretations. Again looking at modalities:

Visual:

Visual disturbances were denied, visual acuity, and extra ocular motion were normal, as were reflexes to light and accommodation. Visual tracking function for saccadic, pursuit and nystagmoid motion was normal, as was opto-kinetic nystagmus, i.e., the purely visual inputs were normal. Absence of oscillopsia or pathological nystagmus and ability to normally track a head synchronized target during S.M.S., argue against other sensory

modalities disturbing visual function, i.e., the visual information should be valid.

Vestibular Function:

Canal function appeared to be normal for while there were changes in VOR gain, as could be determined from eyes occluded head oscillation, the differences appear random in time and between subjects. The strongest evidence for the role of vestibular inputs are the overwhelming conscious sensations which occur with motions. In many, the pitch plane is most sensitive, while in others it is yaw ,but in any event it is a potent sensation. There is no such obvious argument for the static components of the otolith organs except by allowing a priori that this is a cause of S.M.S. If this is allowed then one can argue that the static component must be effective for there are several documented examples of individuals vomiting very quickly on reaching orbit, and before appreciable motion occurred. Also stopping all motion may cause some improvement in feeling, but it does not cure S.M.S., and I have evidence from G.I. studies to support this. Stopping motion

probably only removes the unpleasant sensations from motion and has little objective effect on the underlying process. An outstanding example of this is one subject who simply clung to supporting structure, bat like, with eyes closed for two nights and a day without improvement.

Somatosensory Inputs:

The only direct study of this system was the kinesthetic position sense and eye hand tracking but these did not look at senses which would be most likely to be involved in gravity produced signals, hence it could be argued they are irrelevant. The number of studies during S.M.S. are small and not statistically significant to date, but there are no significant changes in performances during or after S.M.S. One subject was loaded to the equivalent of his own weight by the treadmill harness and stood quietly for a prolonged period without improvement in symptoms.

Visceral Inputs:

We had no way to study this, and other than the gastric symptoms noted, visceral sensation did not reach consciousness.

In summary then we have no evidence for the role of altered or disturbed sensory or neurological systems and considerable evidence against such. At the same time there is a strong theoretical argument for a sensory conflict between the canal and statolith organ signals. This argument is consistent with the phenomena observed. Visual signals are not altered and should be consistent with canal signals both of which conflict with dynamic statolith signals. Visual scenes may produce conflicts with stored information from previous experience or possibly with static information from statoliths or somatosensory signals. The role of somatosensory or visceral inputs is unknown but not obvious.

Neuroanatomy also seems to be consistent with a major role for vestibular conflict since there are known pathways connecting the end organs to the one area which is consistently affected by SMS, the UGI tract. This drawing was constructed from the latest available literature on demonstrated pathways and illustrates not only the intimate physical nature of vestibular and gut nuclei, but the pathways as well.

This brings us to our current problem - ignorance of basic mechanisms. We speak glibly of conflicts but, in fact, haven't a clue of what is really happening. In fact, we don't know the different pathways or the nature of the signals which cause our problems - the ileus of the upper GI tract or the head symptoms. There are two basic possibilities here - neurological transmission and/or humoral transmission. This remains an open question and while I personelly feel that the neurological pathway is more likely, nevertheless I injected naloxone and opioid blockes during S.M.S. (without effect) on my last flight, and collected serum for a search for strange agents.

The question has been raised by one set of experiments of whether the CSF might not be a pathway and this certainly deserves consideration.

Summary:

Space Motion Sickness, now seen from our perspective, is a probable variant of 1g motion sickness with major differences in many aspects. It is a mild form which is not incapacitating to

trained individuals who can still perform demanding tasks with it. It has been universally self limiting, usually clearing within 36 hours and does not recur on continued exposure and appears to be moderated by repeated experience. It appears to produce an upper GI ileus in all those affected and vomiting is secondary to this ileus, not a primary event.

At this time it appears that an intra-vestibular conflict is the primary cause with unknown contributions from other modalities. Since weightlessness cannot be reproduced on earth, there are no good models on earth nor any obvious ways to test for nor acclimate subjects to this conflict. Our knowledge of the neuronal mechanisms involved is grossly inadequate for understanding of the process. We may be lucky and find some breakthrough, some drug or some way to stimulate the conflict on earth, but until then we must study S.M.S. in the only place it occurs, in space. For me the most important aspect of our investigation was demonstration that useful objective data can be gathered quickly and with minimum resources during operational

missions. The frustrating reality is that neither technical problems nor astronaut recalcitrance are the limiting factors. My own opinion is that it may be a decade or more until we cure or prevent either ordinary M.S. or S.M.S. and that will depend upon an understanding of fundamental processes at the neuronal and molecular level and this will be done in labs on earth.

Finally, scientific evidence is determined by scientists, their techniques and interpretation. The life blood of science is diversity and disagreement and I shall be disappointed if differing data and views are not presented here. Only the caustic of time and experience is adequate to prove the correct view and I cheerfully submit these few observations to that process.

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