Sudden Death of Filipino Men in Hawaii*

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DR. ALVIN MAJOSKA published in 1948 in the Hawaii Medical Journal a report of 120 young strong Filipino men who had gone to bed apparently perfectly well and who in the course of the night were heard to groan, gasp, and die. Majoska wrote, "despite all efforts to determine the cause of this syndrome, the initiation of the fatal sequence of events is still unknown . . . The fatal syndrome seems to affect only healthy young Filipino men." This syndrome was first described in the Philippines in 1914.

As to "only Filipino men", Nolasco, in a report of 30 of these deaths in Manila, wrote "26 were in Filipinos, 4 in Chinese." Dr. Yoshimura (Tokyo Police Coroner) reported 157 of these deaths in young Japanese, mostly males, during 1955, '56 and '57.

While I was in San Francisco, Dr. Irma West asked me to review the protocols of 19 autopsies on young Mexican workers who had died in their sleep. Their deaths and autopsy findings were identical with those of our Filipinos. I also have an account of a young Caucasian male, age 20, who died similarly; and also one in a Caucasian woman, age 26. It is evident this syndrome is not limited to Filipinos.

Moritz and Zanchek compiled after World War II the findings of the post-mortems of 40,000 American soldiers. Of these, 1,000 had died suddenly at a time they were believed to be in excellent health. Post-mortem examination revealed one-third had died of unsuspected coronary disease; one-third had died of cerebrovascular accidents; and most of the remaining one-third were signed out as "overwhelming respiratory infection." However there were 140 of these who at post-mortem revealed no cause for death.

Similar studies have been done on over 2,000 cases in Manhattan by Halpern and Rabson. They also reported on a German study of 2,668 with similar results to the World War II study. It is interesting that in each of these series a certain number had "died suddenly" with no cause for death being found at autopsy.

The French and the Germans, during and after World War II, became interested in "sudden death without cause." They came to the conclusion that certain individuals had a hypersensitive mechanism of the vaso-vagal system. Durow and Richet introduced the concept that such sudden deaths could only occur in individuals where the "soil" was previously prepared — that the patient was in a certain sensitized physiological condition when a shock to an inhibitogenic area was received. They wrote, "the reflex was essentially a vegetative reflex and one knows that great variability of these reflexes, not only in one subject to another but in the same subject at one time and another." The French coined a term for this syndrome, "death by inhibition."

They tried to verify this in animals and in the course of some circulatory experiments on 41 cats, the afferent vagus nerve was stimulated. In 31 of these cats no reaction whatever occurred. In nine there was a marked lowering of respiration, blood pressure, and pulse. In one, respiration stopped.

From Missouri, a veterinarian wrote, "As a student of veterinary medicine I am somewhat intrigued by several parallels in the symptomatology of the condition you have described with that of a very interesting and equally baffling phenomena which occurs among the bovine species. Briefly described, this condition appears among animals on full feed and characteristically attacks the most vigorous animals in the herd. . . . The autopsy findings vary greatly. . . . Animals which died suddenly showed few pathological changes. Perhaps only slight petechial hemorrhages in various organs. . . ."

A third animal experiment was reported by a

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hunter who had chased a deer all day. Several times he had almost got it. Toward the end of the day (when the deer was undoubtedly highly frightened) the deer suddenly appeared from behind a clump of trees and the hunter fired. The deer jumped and fell dead. Careful examination showed that only the tip of his tail had been clipped off.

Soma Weiss did an excellent study on "Instantaneous Physiologic Death" and wrote, "some die suddenly with no structural cause and with nothing found at post-mortem." He added, "Increased irritability of the afferent nerve endings on the central synapses produces hyperactivity of these reflexes. The sensitizing factors may be merely emotional or chemical . . . cardiac dysfunction can be induced by stimulation of various reflexes . . . coronary sclerosis gives a tendency to hypersensitivity of the vagal type of carotid sinus reflex as well as other vagal reflexes . . . surgical manipulation of certain nerve structures can be done with safety; in a few cases such stimulation precipitates an alarming seizure or death." These comments on coronary sclerosis might well explain why certain sudden coronary deaths at autopsy show relatively little cause for death and no more coronary atherosclerosis is found than is often found incidentally in people who have died of other causes.

The variation of the sensitivity of the carotid sinus is indicated by an experiment on a series of my own cases. I had two patients who reported they had had attacks of syncope for no known reason. In one of the patients the attack came while he was driving. When he came to he found his car on a strange lawn; a policeman was there investigating his problem. When I rubbed the carotid sinuses of these two patients, they fell into a chair and said they felt on the point of syncope. I stimulated in a similar way 20 other patients and they complained of nothing except the discomfort of the neck rubbing. This type of carotid sinus sensitivity is undoubtedly a rare phenomenon, but it does occur. Death from light trauma has also been reported from several countries.

The areas in the body that have been found to be inhibitogenic are the larynx, abdomen (pancreas) neck of the uterus, testicle, and pleura. Diagnosis can only be made by elimination, i.e. no somatic cause can be found to explain the sudden death, hence "death by inhibition of a vital function." A few case reports will illustrate this phenomenon:

CASE REPORTS

Case 1 A.G. A Sacristan of a small Congregational church caught a small boy throwing snowballs into the window of his church. The boy's father came to his son's aid, struck the Sacristan a blow in the epigastrium whereupon the man fell, tried to rise, and fell dead. He was 44 years of age. Post-mortem was negative; sudden death following light trauma.

Case 2. A jailkeeper, aged 26, received a kick on the throat from a prisoner. He raised his arms and cried, "I am dead," and fell dead. He was a strong man. Post-mortem showed no lesions.

Case 3. A certain night watchman, who loved his wife, came home unexpectedly one night and found a man in bed with his wife. The frightened visitor jumped out of the window, and as his wife was about to follow the husband grabbed her by the throat. With this she collapsed and died. He called in his neighbors, told them the story, but in spite of all efforts the wife failed to respond. A careful autopsy was done; all the organs of the throat were carefully preserved and studied for any possible injury, but nothing was found. Did the excitement of the moment sensitize her vagus? The stimulation from the sudden squeezing of the carotid vagal plexus of her throat stopped her breathing. These cases cannot be explained except by the mechanism of death by inhibition.

In a recent book on syncope, reported in *Today's Health,* Professor Engle showed that most cases of syncope were in males. He also indicated that a person's resistance to such reaction can be increased. He mentioned a study on a group of pilots who had to undergo trials in a decompression chamber. In the first series of tests 18 per cent keeled over. By the seventh test only 2 per cent fainted. None of these healthy men died, but might the mechanism of sudden death be similar to that of syncope, but stronger?

The effect of sleep or drugs on this mechanism has been reported. One experiment was on dogs. Two sets of dogs were given the same dose of morphine. When in a deep sleep one-half of the dogs received a brain irritation by rapidly stimulating the ear drums with a sharp needle. A high percentage of those dogs died, whereas all the dogs without irritation recovered.

Dr. J. B. Nolasco of Manila, showed in some experiments on fish poison that dogs under barbituates died, whereas when the same amount of poison was administered without barbituates the dogs survived.

In the "Filipino dream cases" there were five

reported who recovered and showed no bad after effects. One of these reported by Majoska was of a man sharing a bed with another Filipino. At the first groan the second man awakened his groaning friend, who, sweating and pale said, "Thank God you awakened me. In another minute that little man would have choked me to death." Here we have possible psychic trauma to an inhibitogenic organ (larynx), and the patient recovered with no after effects. Eighty per cent of the Filipinos at autopsy showed marked congestion, often diagnosed as hemorrhagic pancreatitis. We believe this may have been due to the patient having eaten an unusually large meal before going to bed, (a habit of Filipinos) and the congestion of the pancreas was due to the overload of work? The same sized meal could be eaten by the same person during the day without difficulty since only 50 out of 30 million Filipinos per year die of this condition. Hence only relatively few had the hypersensitized mechanism which produced inhibition of the respiratory centers. In addition to the psychic trauma of the dream, perhaps the congestion of the pancreas added to the hypersensitization of the mechanism, thus producing the inhibition in the central respiratory center. That this explanation is not too far fetched is indicated in a report by Dr. Gaskill of England. He reported on 12 cases of unexpected sudden death in apparently well people in which hemorrhagic pancreatitis was the only cause found. Ordinarily acute hemorrhagic pancreatitis, when it is fatal, takes one or two days of agony before death occurs. Some of these deaths were so sudden they must have been due to reflex inhibition. One of his cases was in a 19-year-old student. This man had eaten a heavy lunch and then joined a 'hare and hound' race. After going about one mile he complained of pain in the abdomen and his fellow runners sat him down by the roadside, thinking he was fatigued. When the next group of runners arrived they thought he looked queer. They shook him and found he was dead! Post-mortem showed only hemorrhagic pancreatitis. In these unusual cases death was sudden. Was it inhibition of a vital function in a person who happened to have a hypersensitized vaso-vagal apparatus? In the Filipinos who died and in the Japanese as well as the Mexican workers, the pancreatic lesion was mentioned. In those who dreamed and recovered there was always a dream suggesting sudden death connected with an inhibotogenic region. However, that a nightmare alone does not kill is indicated by the hundreds who have had violent nightmares without serious consequence. Nolasco reported on 20 Filipinos who had had violent nightmares without signs of the Filipino dream death. There must be more than a bad dream in the fatal cases. The plantation doctors who are in charge of the health care of some 10,000 Filipinos reported over a ten year period only 13 of these deaths which suggests the rarity of the fatal reflex. No doubt many ate heavy meals before going to bed and many had nightmares. But if the vaso-vagal mechanism had not been sensitized these experiences failed to produce a reflex stoppage of respiration.

CONCLUSION

We feel justified in assuming that in certain individuals a hypersensitive mechanism of the respiratory center may be present. It can be magnified by psychic trauma, light physical trauma to an inhibitogenic area, or be sensitized in some by coronary atherosclerosis or chemical poisons. In the hyper-sensitized individuals minor trauma or sudden emotional stress can cause cessation of breathing and death. We feel it is a real syndrome which the French called "death by inhibition."

LETHAL EFFECT OF ALCOHOL-BARBITURATE MIXING

The barbiturate drugs used in sleeping pills hinder the body's ability to dispose of alcohol. Because of this interference, the depressant effects of the barbiturate and the alcohol are combined, and what may be merely a sedative dose of barbiturate and a slightly intoxicating amount of alcohol can become lethal. Specifically, the drugs interfere with an enzyme called alcohol dehydrogenase, which controls the first step in the disposal of alcohol. The evidence for these findings came from test-tube studies. Preliminary work with rats points to the same conclusion, and presumably the human body would react similarly.

These findings were reported at the American Chemical Society's Southwest Regional Meeting in December 1963 by Dr. Jack E. Wallace and Elmer V. Dahl of the U. S. Air Force Epidemiological Laboratory, Lackland Air Force Base.