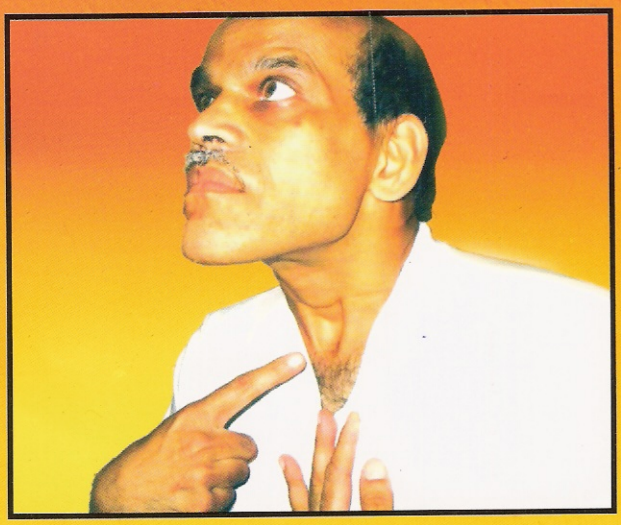
Keep obesity away..
Get rid of gastrointestinal disorders..
Gain capability to float on water..

Drink Air, Stay Fit



काकीमुद्रा-
काकचञ्चुवदास्येन पिबेद्वायुं शनैःशनैः।
काकीमुद्रा भवेदेषा सर्वरोगविनाशिनी।। घे.3/66
Kakachanchuvadasyena pibedvayum shanaih shanaih।
Kaki mudra bhavedesha sarva rog vinashinil। G.3/66

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Preamble

Yoga is increasing in popularity by leaps and bounds. Some proponents claim it to be the panacea of cures for every disease from amoebiasis to Alzheimer's disease and from coronary artery disease to cancers.

Still, since yoga has been brought to us through the television channels, for most of us yoga means performing a few asanas and a few pranayamas. The internal cleansing procedures in yoga exemplified by *vata-sara* have been largely ignored, both due to their complexity and due to lack of medical knowledge in the yoga gurus. Yoga gurus have been scratching their heads to arrive at the correct meaning of the ancient 12-step *Suryanamaskara* (Sun salutation), which has been debated for long.

Air is present in the human gut almost universally. Yet, medical books by and large have ignored its importance. The possible areas of interest being-

1.Obesity (with its resultant problems diabetes mellitus, hypertension and atherosclerosis) and 2.Various gastrointestinal disorders- are two of the major health concerns today, and the author begs to point out that air drinking can be useful in both.

The present work attempts to see relevance in the ancient yogic practice of Suryanamaskara. In the author's view, it is a manoeuvre to fill the intestines with air.

Further, filling of the intestines with air makes swimming much more easy and efficient, so much so that it is only on reading this book and practicing accordingly that swimmers will realize what they were missing till now.

The air taken-in by drinking passes down the gut and is ultimately passed out as flatus. (If someone is afraid of flatulence, my recommended reading is the collection of essays '*Fart Proudly- writings by Benjamin Franklin you never read in school'* edited by Carl Japikse)

CONTENTS

Obesity

While on this earth millions of people are starving about 20% of the population is suffering from obesity. This has become a global epidemic. The hazards of obesity are well known. Every newspaper and magazine carries articles on obesity. It is the forerunner of insulin resistance and diabetes mellitus, and is commonly associated with hypercholesterolemia, coronary heart disease, gallstone disease and osteoarthritis of the knees.

Sleep apnea is another complication in which the affected individual suffers from obstruction to the airway in the throat with resultant interruptions in the nightly sleep. So much can be the disturbance that the individual feels drowsy in the daytime. Such patients are required to sleep with a machine which runs on electricity and provides respiration through mask attached to their mouth and nose when respiratory obstruction develops. So now we have to depend on machines even for breathing during sleep!

After all what is the treatment for obesity? Several medicines have been marketed in the last decade and people used them, but then how long can one go on taking medicines? Then there is fear of adverse effects. It also imposes an economic burden on the individual.

The most commonly suggested way is to eat less and burn more calories by exercise. Is this the only way available or is there an easy option in yoga?In the ancient Indian scripture *Rigveda* there is a mention that in the ancient times the *rishis* and *munis* used to eat air.

मुनयः वातरशनाः पिशंगा वसते मला। (केशी सूक्त, ऋग्वेद, 10,136,2)

'*Munis* eat air, wear cloths of varied colours and stay dusty..'(*Keshi Sukta, Rigveda*,10,136,2.)

Is it possible that we eat less and still feel satiated? How did the *munis* stay comfortable without eating for days at a stretch?

CHAPTER - 1

OBESITY

1.1. INTRODUCTION.

Obesity is acquiring proportions like a global epidemic. Not only the developed countries but also the developing ones are all facing this problem. Obesity is associated with shortening of life span and complications which make life difficult for the individual. In view of this, its prevention, rather than cure must be considered with utmost priority by health authorities the world over.

1.2.1.CAUSES OF OBESITY:

Obesity can occur as a part of a wider systemic disorder, the list of which includes-

(a) Genetic Syndromes: There are some well recognized syndromes which are known by the names of their inventors, such as the 'Prader-Willi Syndrome'(PWS)[1], where there is deletion of some genes on the 15th chromosome or, in some cases, both the chromosomes of the 15th pair are acquired from mother only, the 'Laurence-Moon-Biedl syndrome' etc. Now-a-days great efforts are being done by researchers to precisely identify and locate the defective gene. Much success has already been achieved.

(b) Endocrine disorders: Hypothyroidism, Cushing's disease

(c) Hypothalamic lesions: Damage to a certain part of the brain called the hypothalamus-which regulates appetite and feeding behavior and body weight-is rarely ever a cause of obesity.

All the above systemic disorders collectively make up less than 5% of the obese population. The rest 95% are people who suffer from what is known as 'Simple obesity'.They have no genetic or hormonal abnormality.Their obesity simply results from an imbalance between intake and consumption of calories, or a slight disturbance of appetite and body weight regulation.

It is also of interest that obesity is largely restricted to humans and animals which are domesticated or live in a zoo.

1.2.2.What is the cause of 'Simple Obesity'

Fat is stored by the organisms to be used as a source of energy in times of food scarcity. Hibernating animals store fat before going in to hibernation and use this body fat as a fuel during the cold months. They emerge lean and thin. Non-hibernating animals in most cases, maintain a constant body weight despite cyclic changes in the availability of food. Several studies have been conducted to find out why obesity occurs.

Twin studies[2] have been done to find out importance of genetic and environmental influences.

Experimental studies (in animals): In 1994 a naturally occurring mutant obese mouse was discovered.[3] It was seen to have ob/ob genes. The product of the ob gene is a 16 kda protein (Hormone) called the leptin. The ob/ob mice were found to be deficient in leptin. Injection of leptin in these mice produced satiety and reduced weight. For this reason leptin was also called the body's lipostat. Many hopes were pinned on leptin as a possible therapy for obesity. However, normal animals as well as humans can not be equated with the genetic ob/ob mice. In normal human subjects the leptin

production is proportionate to the amount of adipose tissue present in the body. In obese individuals the leptin levels in blood plasma are already high[4]. For this reason the possibility of leptin deficiency being a cause of common human obesity and leptin being used as an anti-obesity agent have become remote.While leptin functions to maintain the mass of adipose tissue constant, it does not terminate an individual meal on its own. It is thought that it modifies the response to other chemicals e.g. the satiety peptides.

As is obvious, in obese individuals the large amount of fat stored in the adipose tissue, which can serve as a source of energy, does not suppress the desire to eat the daily amount of food. When a person becomes obese, despite the high levels of leptin the appetite continues to be vigorous. Still, rather than giving up on the leptin theory and finding some other chemical (Or its lack) responsible for obesity, some authours started viewing obesity as a case of leptin resistance.

1.2.3.Regulation of body weight:

A part of the brain known as the hypothalamus is involved in regulation of body weight, both in the short and the long run.

Certain areas of the brain-called the hypothalamic ventromedial, dorsomedial, paraventricular and arcuate nuclei are involved in appetite regulation. They are shown to have receptors for leptin. The hypothalamic and other brain areas receive signals from the olfactory and taste receptors as well as from the oral mucous membrane, the jaw and muscles of mastication. These impulses reach the brain via the different cranial nerves, the olfactory, lingual, trigeminal, glossopharyngeal and the vagus. The importance of the filling of the stomach was clearly demonstrated in the earliest

experiments which showed that dogs continue to eat endlessly if the food that they swallow is not allowed to reach the stomach. (Sham feeding)[5]

1.2.3.1. Ghrelin:

The gastro-intestinal tract is actively involved in the regulation of meal size. Hunger has been shown to be proportionate to the strength of contraction of stomach as early as about a century ago by Cannon[5], who also gave us the term 'hunger pangs'. Now it is well established that the stomach secretes a peptide called 'ghrelin' in the fasting state. Injected ghrelin causes hunger. In the filled state of the stomach the stretch receptors get activated and give rise to a sensation of satiety. Also, intake of food inhibits the release of ghrelin. It is this mechanism that is somewhat defective in persons who develop obesity. In obese individuals food fails to suppress ghrelin secretion[6]. Sensation of the stomach being full or empty is carried to the brain via the two vagi. (singular-vagus).

1.2.3.2. Satiety peptides:

After food reaches the intestines, the intestinal mucosa senses its calorie content and in response it secretes several chemicals such as cholecystokinin (CCK), oxyntomodulin and peptide tyrosine-tyrosine (PYY), bombesin, glucagon-like-peptide-1 (GLP1), enterostatin and somatostatin. When injected, several of them are able to induce satiety and reduce food intake. Together they have come to be known as 'satiety peptides' Most of them act on the afferent nerve fibers contained in the vagi, but some also reach the brain via the blood circulation and act on the hypothalamus directly. Some of these also cause secretion of insulin, they are termed 'incretins'.

1.2.3.2.1. GLP-1.

GLP-1 which is a member of the pancreatic polypeptide group has occupied a special position. It is secreted by the enteroendocrine L cells of the small intestine. Physiologically it regulates appetite and is a stimulus for the secretion of Insulin. Orally given glucose releases GLP-1, while intravenous glucose does not. It has been shown that in obese persons the GLP-1 secretion in response to a meal is less than in a normal-weight individual[7]. GLP-1 has a very short plasma half life, being quickly excreted by the kidney and degraded by the enzyme dipeptidylpeptidase-IV. For the purpose of study, it has to be administered by continuous intravenous infusion. Infusion of GLP-1 produces satiety, reduces food intake, increases insulin secretion and in the long run, reduces body weight. In animals it has been shown to promote regeneration of pancreatic ß cells- the cells responsible for insulin secretion. A drug, exenatide[8], a GLP-1 receptor agonist has been developed and is used in type-2 diabetes patients. It has been approved by the US-FDA. It has a longer plasma half - life and can be given subcutaneously. It causes early satiation and reduces food intake, reduces body weight, but also can cause nausea and sometimes a 'feeling of illness'.

1.2.3.3. Other neurotransmitters:

Hypothalamus is also acted upon by several other chemicals such as adrenaline, dopamine, 5Hydroxytryptamine (5-HT), and some cellular messengers called cytokines such as interleukin-1 (IL-1), tumour necrosis factor (TNF) and endocannabinoides. Efforts have been made to develop drugs based on these chemicals, such as sibutramine which is a 5HT agonist. Recently, a cannabinoid receptor-1(CB-1) inverse agonist Rimonabant[9]

has been approved in India and awaits approval by the US-FDA.The clinical results on wider use remain to be seen. Over all, so far the drug treatment remains far from ideal.

The gastrointestinal regulation of food intake has been extensively reviewed by David E. Cummings and Joost Overduin[10]. However, in the author's opinion the commonly held view that 'gastric satiation is volumetric, intestinal satiation is nutritive' endorsed by them is seriously challengeable because of the following:

1. Intestinal distension has not been investigated by any researcher, probably because of the obvious difficulty involved in introducing a balloon in the whole length of the intestines.

2. The role of CCK has been only partially investigated. We must not forget its earliest known function, contraction of the gall bladder. Parenterally administered CCK contracts the gall bladder and the resultant outpouring of cholesterol-rich bile in the duodenum may itself contribute to the satiety.

3. While studying intraduodenal perfusion, corn oil has been used[10]. Intraduodenal (ID) perfusion of cholesterol has not been studied. Vegetable oils do not contain cholesterol.

Recent evidence points to the fact that the cholesterol that is synthesized in the liver and secreted in the bile has several functions in the gut[11,12]. In the author's opinion it is possible that pouring of cholesterol-rich bile into the intestinal lumen due to gall bladder contraction contributes to satiety. It remains to be seen whether intestinal distension by air releases GLP-1. Going by personal experience, air-filling manoevers do lead to a temporary satiety, which may be mediated by GLP-1.