**A Device for the Treatment of Adult
Sleep Apnea Syndrome**

by

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(ABSTRACT)

An electronically-controlled positive-displacement bellows-type air pump has been developed in the Bio-medical Engineering Laboratory for the treatment of adult Sleep Apnea Syndrome (SAS). An electronically-controlled positive-displacement pump has been employed in order to eliminate the pressure regulator and accompanying noise of present therapeutic devices. The positive-displacement pump is found to quietly and effectively provide the required airway pressures for the treatment of adult Sleep Apnea Syndrome. New developments in the reduction of the size and noise levels of current therapeutic devices, however, preclude mass production of the bellows-type pump because of its size disadvantage. The custom nasal mask and exhalation valve, control system, pressure-monitoring alarm system, and the controlled humidifier of the positive-displacement pump should be incorporated within the present fan-type Nasal Continuous Positive Airway Pressure (NCPAP) system to provide quieter, more comfortable, and safer NCPAP therapy. Before the design and development of the positive-displacement NCPAP pump is detailed in this thesis, however, the problem of adult Sleep Apnea Syndrome is introduced, available therapies are investigated, and the current NCPAP therapy system is examined.

Acknowledgements

I would like to express my sincere thanks and appreciation to the following people and note their contribution to this research.

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generously contributed her time and NCPAP device. Without her assistance this project would not be complete.

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Finally, all my family and friends held their belief in me and showed constant interest. Someone always had a joke and words of encouragement -- even at the lowest ebb. For this, I am greatly indebted.

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Glossary

Apnea   The periodic cessation of breathing

Apnea Index   Severity measurement of Sleep Apnea Syndrome meaning the number of apneic events per hour

Bradycardia   Condition marked by slow heart action

Cardiac Arrhythmias   Irregular heartbeats

Cor Pulmonale   Enlargement of the right heart

Enuresis   Incontinence, uncontrollable urinary release

Hypnagogic   Induced by the drowsiness just prior to sleep

Nares   Nostrils, nasal passage

Necrosis   The death of tissue in areas surrounded by healthy tissue

Pharynx   The throat, the muscular membranous tube extending from the cavity of the mouth to the esophagus

Phrenic nerve   The pathway for electrical stimulation of the diaphragm

Pulmonary Edema   The swelling of the lungs caused by the leakage of fluid from the blood into the aircells (aveoli) of the lungs

Rapid Eye Movement (REM) Sleep   The sleep stage most associated with sleep apnea due to the loss in muscle tone

Sleep Apnea Syndrome   The condition marked by the periodic cessation of breathing for longer than 10 seconds during sleep

Glossary

Stenosis   Constriction or narrowing of a passage or an orifice

Tidal Volume   Normal respiration volume of a healthy resting adult

Uvula   The pendent fleshy lobe found in the middle of the border of
the soft palate

Introduction

Sleep Apnea Syndrome (SAS) is a potentially life-threatening disorder affecting anywhere from 0.1% to 4% of the population. As shown by the wide range in estimated incidence, the occurence of this recently recognized disorder is not known exactly. It is believed that between 200,000 and 2 million sufferers may reside in the United States [1,2][*]. Although SAS is most prevalent in 40 to 60 year old overweight males, the malady is not restricted by age, sex, or body type.

Sleep Apnea Syndrome is the periodic cessation of breathing with a duration over 10 seconds during sleep. Not known exactly, the mechanism of an apneic event is thought to involve the loss of muscle tone in the upper airway during sleep and the need for large subatmospheric pressures during inhalation for people with slight upper airway disturbances. Alone or with possible fluctuations in respiratory control during sleep, these conditions may lead to the blockage of the trachea by the tongue and surrounding tissue. This leads directly to an apneic episode.

Some sleep apnea patients suffer five to six hundred episodes of apnea during a single night's sleep and suffer extreme fatigue from the resulting sleep deprivation [2,3]. One individual with SAS was recorded to have

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Note: Numbers enclosed in brackets refer to cited references found at the end of this thesis.

stopped breathing for a total of 3 1/2 hours in an 8 hour sleep [4]. With durations of 10 to 120 seconds these apneic episodes may cause an individual's blood oxygen saturation level to dip down to 30%. For comparative purposes, blood oxygen saturation levels in the range of 85 to 90% are considered on the low end of the normal range.

Chronic episodes over months and years may lead to serious cardiac arrhythmias (irregular heartbeats), generalized cardiac enlargement, congestive heart failure, cor pulmonale (enlargement of the right heart), pulmonary and systemic arterial hypertension, arterial blood gas abnormalities, respiratory failure, and pulmonary edema. Clinical features of the recurring occlusion of the upper airway during sleep may be excessive daytime sleepiness, heavy snoring, restless sleep, morning headache, personality changes, intellectual deterioration, insomnia, sexual dysfunction, hypnagogic hallucinations, and nocturnal enuresis (incontinence) [1,2,3,5].

Sleep Apnea Syndrome may be treated by behavior modification, drug, surgical, or mechanical therapies. Presently, the long-term effectiveness of behavior modification therapies is not clear. In addition, there are formidable disadvantages to the use of drug and surgical SAS treatments. Mechanical treatment of SAS appears to provide the most effective, non-invasive therapy. Current mechanical devices do create auxiliary problems though. The fan-type Nasal Continuous Positive Airway Pressure devices used today are objectionable as a SAS therapy because of (1) the excessive noise of the blower, (2) the uncomfortable and leaky patient interface, and

(3) the dryness of the pressurized air. The new electronically-controlled positive-displacement NCPAP therapy system addresses these problems.

The research work performed in the Bio-medical Engineering Laboratory and detailed in this thesis proves in practice that an electronically-controlled positive-displacement pump will fill the need for a simple, non-invasive, reliable, and effective device for the treatment of adult Sleep Apnea Syndrome. The following research work is based on the pioneering Nasal Continuous Positive Airway Pressure (NCPAP) work performed by Dr. Collin Sullivan et al. [6]. Using this therapy technique as a basis, Dr. Leon J. Arp has developed the original concept for a successful bellows-type positive-displacement NCPAP device.

In this thesis, the construction and testing of a working prototype device is presented. Because of new developments in the reduction of the size and noise of current therapeutic devices, however, mass production of the positive-displacement pump is not practical. Auxiliary systems of the new pump which increase the comfort, effectiveness, and safety of current fan-type NCPAP systems will be detailed. Before this is done however, it is first necessary to develop a firm understanding of fundamental respiratory anatomy and the condition of Sleep Apnea Syndrome. Following this, the treatments and therapies for SAS will be examined with specific emphasis given to Nasal Continuous Positive Airway Pressure therapy and the current NCPAP systems. Finally, recommendations and conclusions about the suitability of such a system in treating adult SAS patients are drawn.

Respiratory Anatomy

The human respiratory system is responsible for the exchange of oxygen and carbon dioxide. Oxygen is a necessary input to the metabolic process while carbon dioxide is a byproduct of the metabolic process. The primary organs involved in the respiratiory process are: the brain, chemical and neural afferent sensors, the physical air passages, the muscles responsive to the neural commands, and the lungs which are the site of the gaseous transfer.

The medulla oblongata section of the brain is responsible for the fundamental breathing rhythm. Various chemical sensors monitor the pH and the partial pressures of carbon dioxide (CO_2) and oxygen (O_2) in the blood and vary the rate and depth of breathing to control the blood chemistry and minimize the body's respiratory work input. Auxiliary neural sensors also provide input for respiratory control.

Air is drawn into the body by the natural negative pressure pump, the diaphragm. An electrical signal from the spine to the phrenic nerve initiates the downward flex of the diaphragm. With this movement the volume of the throracic cavity is increased and a subatmospheric pressure is produced in the lungs. Air then flows into the lungs because of the pressure difference between the atmosphere and the lungs.

Figure 1 is a drawing of the normal adult upper air passage [3]. It is intended to introduce the names of the upper airway features and illustrate the structure of the area. The external nares, or nostrils, are the primary air intake for human ventilation. Although providing greater resistance than the mouth for the flow of air, the nasal passage is a superior intake. The large surface area of the internal nares allows for remarkably effective humidification as well as heating or cooling of the incoming air. In addition, the tortous path and fine hairs of the nasal cavity filter the air before it reaches the pharynx or throat. Finally, the olfactory characteristics of the nasal passage add a quality control meter to the air passage. Noxious or irritating contaminents may irritate the nasal mucosa and initiate a sneeze to reject the unclean air and to protect the passage and lungs.

After passing through the internal nares, the air is diverted close to 90 degrees into the nasopharynx. The sharp turn of the airway provides further protection against the inhalation of large foreign particles. These particles tend to impact the back wall of the nasopharynx and stick to the mucous membranes. Air inhaled through the mouth meets that inhaled through the nose at the base of the tongue or oropharynx. Inhaled air then proceeds past the epiglottis, the tracheal guard for esophageal matter, and into the trachea on its way to the lungs.

Since there is only a single passage to the lungs, both oxygen-rich inhaled air and oxygen-depleated exhaled air must follow the same route. This design produces a respiratory dead-space, a residual volume, which

FIGURE COURTESY OF MEAD JOHNSON LABORTORIES
INHALATION THERAPY HANDBOOK
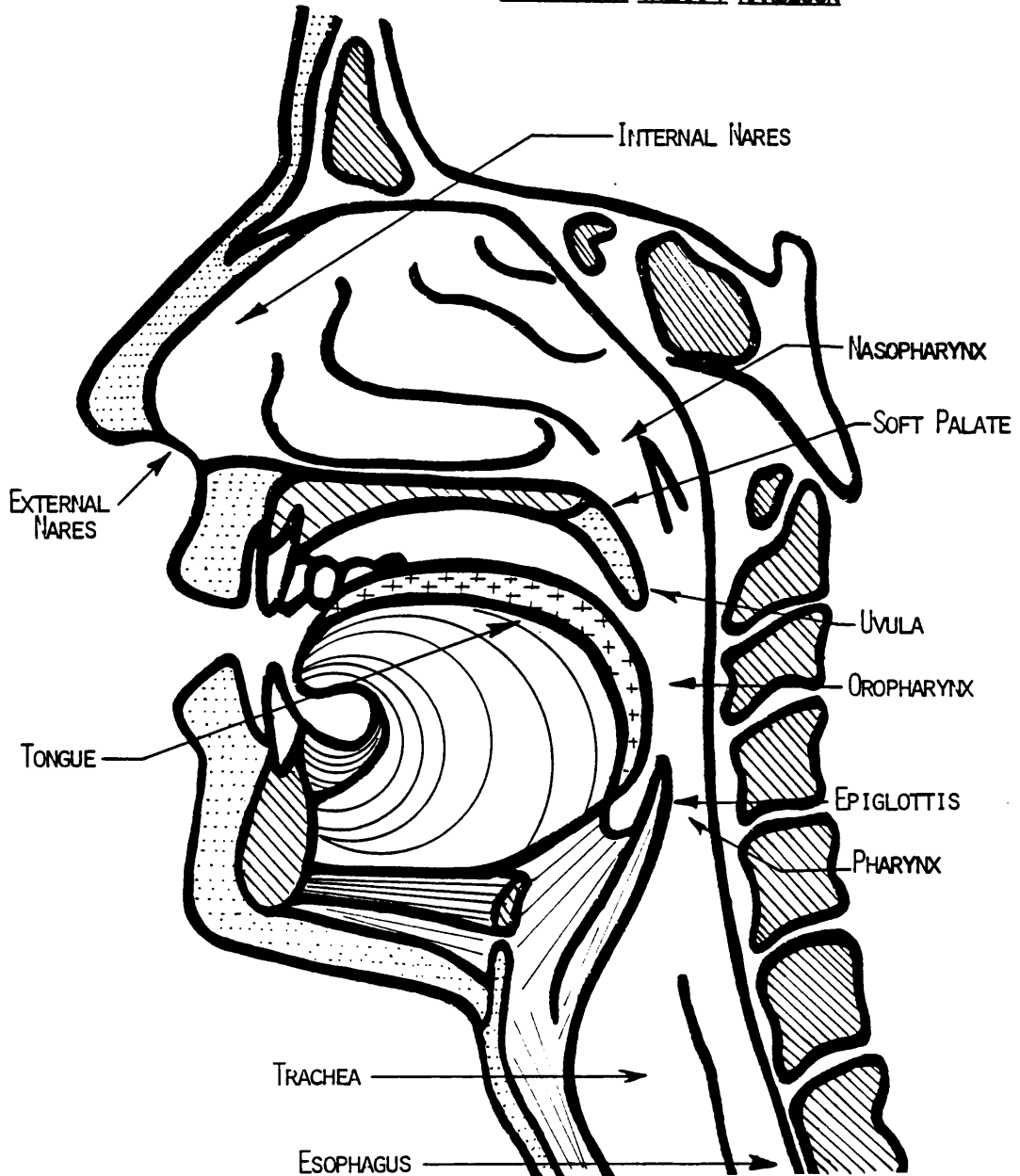
FIGURE 1. ANATOMY OF THE NORMAL ADULT UPPER AIRWAY

unproductively adds to the total respiratory volume. Thus, with even full respiration some exhalation gases remain in the respiratory tract and become mixed with the fresh gases of inhalation. Some common respiratory capacities for a healthy resting adult are pictorially defined in Fig. 2. As shown, the normal lung and trachea has a total volume of approximately 5500 ml with a tidal volume of only about 400 ml to 500 ml. The tidal volume is that volume that is normally exchanged during resting respiration. Since the body at rest requires the transfer of approximately 200 to 250 ml of oxygen per minute, a respiration rate of 12 to 15 breaths per minute is considered normal. Figure 2 shows that in the resting adult there is in excess of 2000 ml of lung volume for additional inhalation and nearly 1250 ml of lung volume for extra exhalation. The residual volume of the normal lung ranges from 1000 to 1500 ml. The vital capacity is defined as the maximum volume of air that one can naturally exhale after maximum inhalation.

FIGURE 2. NORMAL ADULT LUNG CAPACITIES

Sleep Apnea Syndrome

Apnea may be defined as the absence of breathing, but it is usually reserved for that absence of greater than 10 seconds. Sleep Apnea Syndrome, likewise, is defined as the condition marked by the periodic cessation of breathing for over 10 seconds during sleep. A patie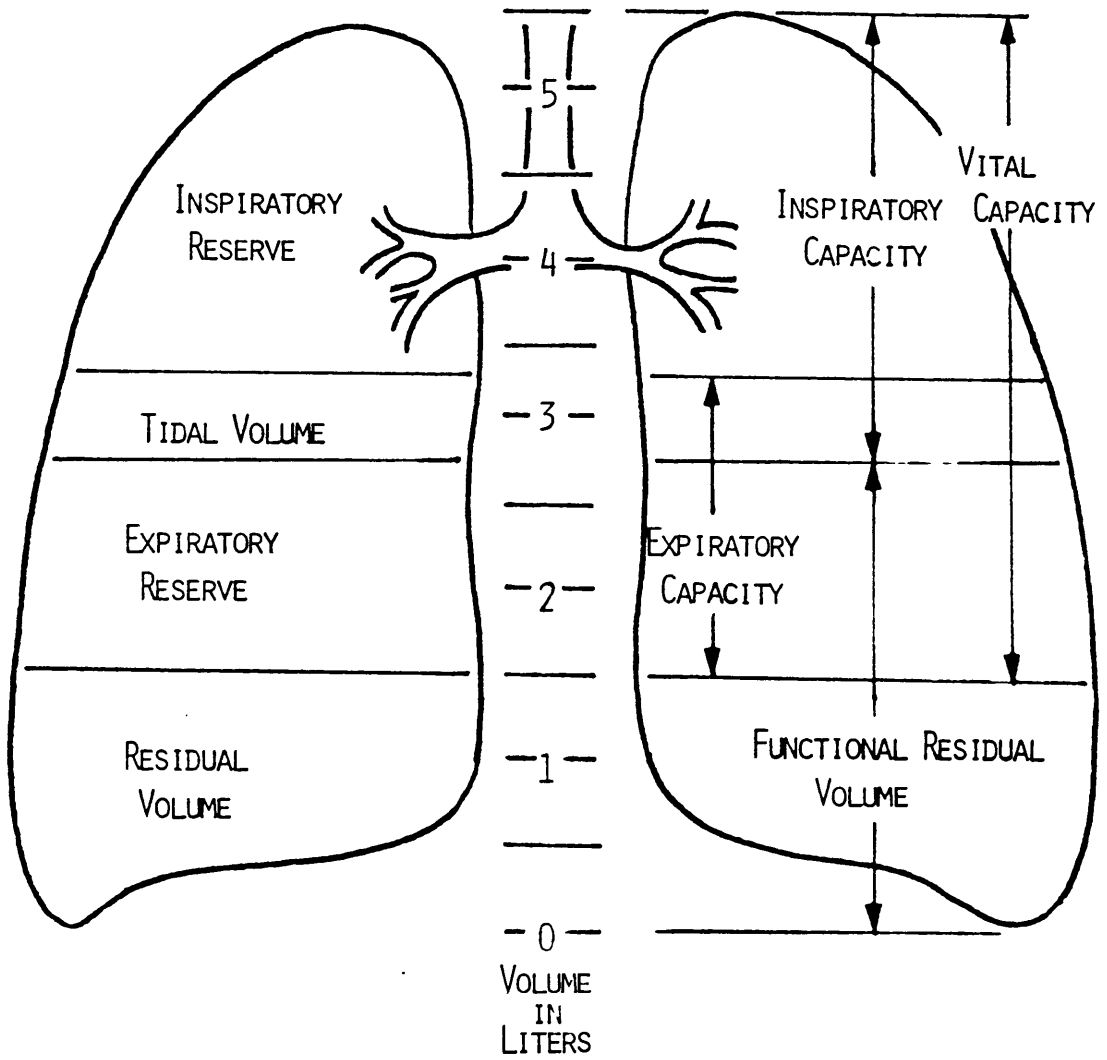nt suffering over 30 apneic episodes in a single 7-hour sleep or with an apnea index, events per hour, of greater than 5 is diagnosed as having SAS. It must be kept in mind however, that age plays a role in the diagnosis of sleep apnea as an apnea index of 5 in an elderly person may be termed "normal".

There are three types of sleep apneas: central, obstructive, and mixed. Central Sleep Apnea (CSA) is characterized by the absence of ventilatory efforts caused by the failure of the respiratory center of the brain to initiate respiration. Obstructive Sleep Apnea (OSA), as its name implies, is characterized by an obstructed airway. Despite persistent ventilatory efforts, there is no respiration. It results from an upper-airway obstruction most commonly caused by the collapse of the pharynx. Mixed Sleep Apnea (MSA) is the result of the combination of factors causing both CSA and OSA. Generally, MSA begins with a central pattern and gradually evolves into an occlusive pattern. It is truly rare when a patient is found to exhibit only a single apnea type. More commonly, a patient exhibits the characteristics of all three apnea types but has a single prevalent type. The predominance of a single type of apnea usually determines the classification.

Although not completely understood, the mechanism of an obstructive apneic event is thought to involve the loss of muscle tone in the upper airway during sleep and the need for large subatmospheric pressures during inhalation for people with slight upper airway disturbances. This purely mechanical obstruction is believed to occur because of the sudden decrease in upper airway muscle tone, loss of stiffness, at the onset of sleep. Some other proposed mechanisms for the disorder cite neural or chemical sensor malfunction as the original cause for the apneic event. These mechanisms propose that abnormal airway mucosal sensations lead to disproportionate or delayed respiratory muscle contraction [2,8], normal mucosal sensations are mishandled by malfunctioning central neural pathways resulting in diminished respiratory neural drive [9], or changes in respiratory control during sleep cause unstable oscillation in blood-gas chemistry which ultimately lead to periods of disordered breathing and a reduction in respiratory drive [10].

It is generally believed that OSA and CSA are alternative manifestations of a single underlying pathology with their true relationship still hidden. CSA patients are less likely to display the clinical symptoms of Sleep Apnea Syndrome, however. These patients tend to be older, have other neurological problems, experience far less oxygen desaturation, and tend to be more normal in body weight than OSA suffers. Because of these differences in the CSA and OSA patients, the following discussion will refer primarily to obstructive apnea (OSA) when refering to Sleep Apnea Syndrome.

Many Obstructive Sleep Apnea sufferers have pre-existing upper airway obstructions. This is supported by the results of a survey which showed that between 70% and 90% of adult sleep apnea sufferers have snored since childhood [2,11]. Snoring, a common occurence in roughly 20% of the general population, can be associated with OSA since alcohol and weight gain may convert a snoring patient into one with obvious obstructive apnea. Alcohol alone may also increase apneas. Alcohol's ability to selectively depress the neural activity of the upper airway dilator muscles reduces the pressures required to cause upper airway collapse by 25% to 50%. Alcohol may also promote apneas by adding to the congestion of pharyngeal mucous membranes and reducing the chemical ventilatory drive [5]. The muscular depression that occurs during sleep and with the consumption of alcohol along with the increased pharyngeal resistance found in snorers may prove to be important factors in the development of SAS.

Airway abnormalities observed during a physical exam of an obstructive apneic patient tend to support the theory that a predisposition for airway obstruction appears in the patient. A physical exam may reveal: a short thick neck with a large uvula, redundant pharyngeal mucosca, enlarged tonsils, bulky elevated tongue or posterior displacement of the tongue, nasal obstruction, and jaw deformaties. Pharyngeal cross-sectional areas for snorers with SAS are comparable with pharyngeal cross-sectional areas for snorers alone, yet are consistently smaller than those of non-snorers at functional residual volume. The reduction in pharyngeal cross-sectional area that occurs during natural resting respiration is larger for apneic than non-apneic

snorers. There is disagreement on whether the key issue is pharyngeal cross sectional area or pharyngeal stiffness, however. Some propose that pharyngeal stiffness is the key factor in determining whether a snorer has SAS or not [12].

Snoring, although by itself not a signal of sleep apnea, may be an early indicator of the sleep disorder. The most common clinical symptoms of SAS are: chronic daytime fatigue, heavy snoring, abnormal physical movements during sleep, diffuse morning headache, severe or rapid personality changes, and intellectual deterioration. Chronic daytime fatigue results directly from the extended periods of light or disturbed sleep. It is common for an OSA patient to experience 30 to 40 seconds of obstructive apnea before 10 to 20 seconds of arousal from sleep with each apneic event. Excessive daytime sleepiness may seriously affect the quality of an employee's work and directly lead to the dismissal of SAS sufferers. Unlike narcoleptics who may feel refreshed after daytime naps, those with SAS tend to experience greater fatigue and fogginess following naps. A recent study demonstrated that subjective sleepiness was most strongly, though not exclusively, related to nocturnal mean arterial oxygen saturation [13]. Heavy snoring, as stated previously, is a common complaint by an overwhelming majority of apneic patients. Abnormal physical movements during sleep, including sleepwalking and sleeptalking, are common in over 50% of apnea sufferers. Recurrent frontal morning headaches that dissipate after several hours and sometimes reoccur after sleep episodes during the day are a common complaint. Along with snoring this is considered a major diagnostic symptom of SAS. Some

common personality changes exhibited by sleep apnea patients include: paranoia, irritability, depression, suspicion, hostility, aggressiveness, and jealousy. In addition, morning confusion and hypnagogic hallucinations may accompany noticeable intellectual deterioration [2,5,11,14]. Some less common symptoms of SAS are insomnia, nocturnal eneuresis, and sexual dysfunction. One half of the patients in one apnea survey report a progressive loss of sexual potence and drive, and difficulty attaining erection and ejaculation [2].

Medically, the most serious result of the sleep disruption seems to be the severe fluctuation in blood oxygen saturation. Saturation below 60% may directly result in serious cardiac arrhythmias. Chronic episodes over months and years may lead to cardiac and pulmonary disease. One study assessing the risk of the most serious effects of SAS found that non-fatal bradycardia of less than 30 beats per minute and sinus pauses of between 2 and 13 seconds occur in 7% to 11% of Sleep Apnea Syndrome patients [13]. Further, dangerous ventricular ectopic beats are found in 1% to 3% of the patients. Cor pulmonale and clinical right heart failure are present in 10% to 15% of patients. This study leaves open the association between OSA and systematic hypertension, however, calling for increased research on this relationship. Finally, the increased stroke risk of heavy snorers is recognized.

Even if the severe cardiac effects of reduced oxygen saturation are not present in the SAS patient, treatment of the disorder should be based on the potentially dangerous and disabling excessive daytime sleepiness found in SAS patients [13].

Sleep Apnea Syndrome Therapies

Sleep apnea treatments range from simple behavior modification to advanced surgical techniques. Factors predisposing to SAS should be corrected first and more vigorous treatment or therapy reserved for more stubborn cases. If obesity is deemed a major contributor to the condition, a strict plan of weight loss should be instituted. However, less than 50% of obstructive sleep apnea cases are caused by obesity [1]. It should be noted that the chronic fatigue associated with sleep apnea is not conducive to a regimented weight loss program and makes this behavior modification therapy especially challenging. If alcohol or antihistamines play a role in the condition, their use must be restricted. Enlarged tonsils or adenoids should be removed. Deformities of the jaw or treatable nasal obstruction should be corrected with reconstructive surgury. Further therapeutic approaches to the management of Sleep Apnea Syndrome resort to the use of pharmacologic agents, more invasive surgical techniques, or mechanical devices. Each of these therapeutic approaches will be examined in the following sections.

Drug Therapies

The magic pill that eliminates apneic episodes is not available. As a result, drug therapy may follow three general categories: those that increase upper airway patency like nasal sprays, those with respiratory-stimulating effects like acetazolamide or progesterone, or those with more general

neuroactive effects like antidepressants [11]. One such antidepressant, the drug Protriptyline, is tolerated well in the long term by approximately one half of those prescribed the drug [15]. Although the drug may reduce apneic events, abnormal sleep characterized by many apneic episodes may still occur. The drug is successful in reducing Rapid Eye Movement (REM) sleep, the sleep stage where most apneic events occur because of the loss of muscle tone. Long-term effects of the reduction of REM sleep have not be established to determine if this is a viable method for controlling SAS. The drug is also successful in reducing the patient's feelings of listlessness, which may help support a weight loss program if this is deemed a problem. There are bothersome side effects to this therapy, though. Dry mouth, urinary retention, constipation, and impotence have all been recognized as significant side effects to the drug. All in all, the effectiveness of drug therapy for the Sleep Apnea Syndrome patient has not been thoroughly established.

Surgical Therapies

The primary surgical techniques used to treat SAS include: tonsillectomy and adenoidectomy for fairly minor airway obstruction, the repair of a cleft palate and deviated septum to reduce airway resistance, uvulopalatopharyngoplasty (UPPP) and palatopharyngoplasty (PPP) for more severe obstruction, and finally a tracheotomy for the most severe and life-threatening disorders.

UPPP and PPP are relatively new and simple surgical techniques that have proven to be successful in about 50% of OSA patients [5,15]. UPPP is the surgical enlargement of the pharyngeal airway by the resection of excess mucosa. PPP is a similar surgical technique differing only in the site of obstruction. The objectives of UPPP/PPP surgery are to separate the posterior aspect of the soft palate from the posterior aspect of the pharyngeal wall, to increase the pharyngeal diameter, to gain space by excision of the uvula and tonsils, and to create permanent non-collapsing nasopharyngeal and oropharangeal airways [2]. While performing the UPPP surgery, the surgeon also installs a temporary tracheostomy adapter to test the improvement. Because there is no effective predictor for success of the UPPP/PPP surgery, the tracheotomy is performed as a backup in case the therapy fails. In the event that the resection alleviates the apneic episodes, the tracheostomy adapter may be removed. This therapy does not come cheaply, however; a uvulopalatopharyngoplasty may cost about $6000. In addition to the high financial cost, patients must consider additional costs, possible surgical complications, when considering this surgery. Some complications to consider are: nasal speech, palatal stenosis, dry mouth, and possible recurrence of snoring or apnea with weight gain.

The surgical tracheostomy therapy is reserved for the severest cases of SAS when all other therapies have failed. It is used for the therapy of both obstructive and mixed apneas because it creates a bypass around the area of critical pharyngeal obstruction. In adults a 6 mm diameter tracheostomy adapter is inserted in the semipermanent tracheal stoma just below the

tracheal obstruction. Before retiring each night the patient opens the adapter and bypasses the resistance of the upper airway during sleep. Upon awakening the patient will cap the stoma for easier speech and cosmetic reasons. Complications of a tracheostomy include: the danger of anesthesia during the surgery, the surgical difficulty in performing the procedure in obese patients, stomal infection, accumulation of granulation tissue around the stoma, the build-up of secretion and an increasing cough, and the inconvenience of meticulous cleaning and positioning of the tube [3,11,15]. Because the tracheostomy adapter is deemed cosmetically offensive to some, this therapy is not for all SAS sufferers. The physician must adequately weigh the personal wishes of the patient with the medical benefits of the therapy when proposing this alternative. The initial cost of a tracheostomy begins at approximately $4000 [1].

Mechanical Therapies

Other therapies available for the treatment of adult Sleep Apnea Syndrome are nasopharyngeal intubation, low-flow nocturnal nasal oxygen therapy, tongue-retaining devices, nocturnal position and alarm systems, and Nasal Continuous Positive Airway Pressure (NCPAP).

For some patients nasopharyngeal intubation may relieve them of the apneic episodes. This procedure, the insertion of a tube through the nares and down the pharangeal airway past the oropharyngeal area, may relieve the upper airway obstruction caused by the tongue's falling back and occluding

the pharynx. This therapy is not tolerated by some SAS patients who complain of its invasiveness and the discomfort associated with the irritation of mucous membranes. Proper hygiene and lubrication of the nasopharyngeal tube and nostrils are important aspects of the success of this therapy.

Studies on the effectiveness of low-flow nocturnal oxygen therapy for the treatment of SAS have been inconclusive [1]. Initial observation revealed that oxygen administration prolonged apneas by inhibiting the arousal mechanisms and reduced respiratory drive. This resulted in acute respiratory acidosis because of the patient's high CO_2 retention. More recent studies, however, suggest that oxygen therapy may reduce sleep apnea and sleepiness in a subset of apneic patients [10,16]. It has been shown that an apneic threshold of partial pressure of CO_2 in the blood exists below which apneic events occur. With a low partial pressure of O_2 brought about by sleep as the initial driving event, the patient begins to increase breathing rate and depth and quickly hyperventilates. The driving effect of the low partial pressure of O_2 predominates over the inhibitary effect of a slightly reduced partial pressure of CO_2. With a final greatly-augmented inhalation, the partial pressure of CO_2 in the blood drops below the apneic threshold rapidly and respiratory drive is severely diminished, causing the apnea. This chanin of evensts appears to have the greatest effect on those patients showing a marked gain in respiratory control sensitivity during sleep to a low partial pressure of O_2 and rising partial pressure of CO_2 [10]. Presently these patients can not be clinically distinguished and more research must be conducted before low-flow oxygen therapy can gain wide acceptance.

Tongue Retaining Devices (TRD) are simple devices that can be fitted to the patient in a physician's office and appear to be a useful first step in the management of SAS. The retaining device holds the tongue in the forward position by suction and prevents it from falling back on inhalation and occluding the airway. It has been shown to significantly improve the sleep of some apneic patients, reducing the number and length of apneic episodes. This therapy is most successful with position-sensitive apneic patients and requires a patent nasal airway.

Nocturnal position monitors may be found to be an effective non-invasive treatment for many apneic patients. One study of 184 unselected patients showed that over 60% of the apneas were position sensitive [17]. It was found that the apnea rate could be cut in half by forcing the patient to sleep on his side rather than his back, the lateral rather than supine position. Although this is a vast improvement it should be noted that this therapy, in general, will not eliminate all apneic events. The combined effects of nocturnal position monitoring and HCPAP therapy may be a significant advancement in SAS therapy, though. Each has been found to provide statistically significant changes in pharyngeal areas [12]. The supine sleeping position was determined to produce a 21% reduction in pharyngeal cross-sectional area from the sitting position, hence the lateral sleeping position is preferred. Use of a CPAP therapy of 5 cm of H_2O while in the sitting position increased the pharyngeal cross-sectional area by 45% from the sitting position alone.

Dr. Collin Sullivan first proposed the use of Nasal Continuous Positive Airway Pressure therapy on SAS patients in 1981 [6]. Since that time many have supported and expanded upon his work. NCPAP therapy is now recognized as a highly effective method of treating OSA patients. Dr. Sullivan also studied NCPAP as a treatment for CSA patients and found that higher NCPAP airway pressures may also be used in effectively managing this condition [18].

By supplying the patient with a continuous positive airway pressure, Sullivan found that the airway remains patent because of a "pneumatic splint" effect. He proposed that each person has a threshold pressure at which a lower airway pressure will cause collapse of the airway. By studying the threshold pressures of non-snorers, snorers, and obstructive apnea patients, Sullivan proposed NCPAP therapy as a treatment for SAS. In his study he found that non-snorers were able to resist an average pressure of -15 cm H_2O, while habitual snorers were able to resist a mean pressure of only -5 cm H_2O before airway collapse. Further, obstructive apnea patients experienced airway collapse at less than -3 cm of H_2O pressure and often needed positive pressures to assure patency [19]. NCPAP therapy is used to raise the inner-airway pressure above the patient's individual threshold pressure, thus preventing airway collapse.

Because NCPAP therapy is non-invasive, presents no pharmacologic side effects, is less expensive than surgery, and achieves instant results, it appears to be a strong first-choice therapy for treating SAS. Some disappointing

features of the therapy are the noisy cumbersome apparatus and the rather low long-term compliance of about 70%. Since the bulk of the research performed in the Bio-medical Engineering Laboratory was in the development of a new NCPAP system, NCPAP therapy and the current devices used to treat SAS will be discussed in detail in the following chapters.

Nasal Continuous Positive Airway Pressure Therapy

Nasal Continuous Positive Airway Pressure therapy may be used as a temporary treatment until surgical treatments become available, acceptable, or effective; as an interim treatment to help support a weightloss plan where the treatment will be discontinued at a certain weight goal; or it may be employed as the primary treatment of choice. It has proven to be effective in about 80% of most populations, but the long-term patient compliance and effects are not yet clear. NCPAP therapy is most effective in treating apneas brought about by the collapse of the oropharynx. Although NCPAP therapy has proven to be an effective therapy for some SAS patients, it is not recommended for all apnea sufferers. Patients with a history of bullous lung disease, pneumothorax, pathologically low blood pressure, severe cardiac arrythmias, seizures, strokes, coronary artery disease, or sinus or middle ear infections should not be prescribed the NCPAP therapy.

Increased intrathoracic pressures may cause a decrease in venous return. Adverse cardiac effects of the therapy were thought to include a decrease in cardiac output, impaired renal function, and an increase in morbidity and mortality [20]. As a result of this, NCPAP therapy is limited to airway pressures below 20 cm H_2O where the cardiac effects of NCPAP therapy are believed to be very low. One 15-patient study found that Continuous Positive Airway Pressure (CPAP) therapy below 20 cm of H_2O pressure actually improved blood oxygenation and vital capacity, and lowered the patient's mean arterial pressure and the pulmonary vascular resistence [20].

Respiratory effort is reduced with CPAP therapy because less change in intraplural pressure is required to provide tidal breathing. CPAP therapy may actually decrease the work of breathing. An experiment on 9 athletes using a therapeutic pressure of 20 cm H_2O showed that CPAP decreased total work per minute by 45% while also increasing the functional residual volume of respiration [21]. The athletes quickly learned to allow the CPAP device to inflate the lungs and expand the thoracic muscles, eliminating the body's work of inhalation. The recoil of the expanded thoracic muscles then aided in the work of exhalation, producing an overall lowering of the net respiratory work. This muscular expansion effect is also credited with increasing the functional residual volume of the CPAP patient.

Mechanisms

NCPAP therapy prevents collapse of the upper airway by acting like a pneumatic splint to keep the airway patent. It was also thought that this therapy may increase upper airway muscle tone directly [3]. The applied pressure was believed to lead to stiffer upper airway muscles which may withstand increased subatmospheric pressures before collapse. This theory is weakened by the sleep study results of non-compliant NCPAP patients, however [23, 24]. These patients occasionally take a night off from therapy. Although they attest to feeling well-rested, these patients are found to have reverted back to their apneic sleeping pattern while temporarily abstaining from the therapy. In addition, there is no evidence to support the theory that NCPAP pressure therapies between 13 and 15 cm H_2O themselves

increase upper airway muscle activity. It is now generally believed that constant positive airway pressure passively opens the airway, by the pneumatic splint method, but further studies may be needed to isolate the specific mechanism of NCPAP therapy.

Testing

Even though the mechanism may not be fully understood, NCPAP therapy has proven to be an effective treatment for sleep apnea. One study of 3 patients with severe sleep apnea, apnea indices ranging from 35 to 63, showed that NCPAP therapy improved oxyhemoglobin saturation considerably [24]. Prior to therapy blood gas analysis showed that hemoglobin oxygen saturations ranged from 90% to 93% during wakefulness, 70.7% to 81.8% during non-REM sleep, and 51% to 71.5% during REM sleep. With NCPAP therapy the oxyhemoglobin saturation rose to 92% to 95.5%, regardless of sleep stage. Consistent with previous NCPAP testing that showed no detrimental hemodynamic effects from the therapy, the same study also monitored mean pulmonary arterial pressures during therapy and found the patient's blood pressure dropped within the range of 20 mm Hg to 20.7 mm Hg, independent of sleep stage. Prior to treatment pulmonary arterial pressures ranged from 14 mm Hg to 20 mm Hg during wakefulness, 16.2 mm Hg to 30.4 mm Hg during non-REM sleep, and 25.1 mm Hg to 46.9 mm Hg during REM sleep.

Further tests were done to prove the effectiveness of NCPAP therapy. One such test was conducted on 11 adult male sleep apnea patients found to successfully respond to the therapy on a trial sleep study [25]. Prior to NCPAP therapy these men were monitored and found to have a mean apnea index of 35.95 with an average 26.68 second duration. They were observed to experience episodes of additional disordered breathing an average of 19.25 times per hour, each lasting an average of 23.1 seconds. During apneic episodes the mean decrease in arterial oxygen saturation from wakefulness was 11.2%. NCPAP therapy between 7.5 and 15 cm of H_2O pressure eliminated all apnea and disordered breathing episodes. A blood-gas analysis revealed that no oxygen desaturation was observed during sleep while the NCPAP system was employed.

Not only was the therapy found to eliminate the breathing disorders, but it was also found to totally reshape the make-up of the patient's sleep pattern. The light sleep of stages I and II that previously accounted for 86.95% of the sleep time accounted for only 52.6% of the sleep time while on NCPAP therapy. This light sleep was replaced with the deep sleep of stages III and IV which improved remarkably. Prior to therapy stages III and IV sleep accounted for an average of 1.5% of the sleep time, while during therapy this increased to an average of 30.9% of the total sleep time. It is not yet known if this drastic increase in deep sleep occurs as a temporary "rebound" effect from prior deprivation, or remains as a lasting reorganization of sleep patterns.

A more extensive 60 patient study was carried out to verify the effectiveness of NCPAP therapy in reducing apneic episodes and restructuring sleep [26]. Of the 60 consecutive OSA patients, 43 (71%) were effectively treated on the trial night with a NCPAP system and their apneic episodes totally eliminated. Before the treatment, a sleep study of these patients revealed a mean apnea index of 76 episodes per hour and a mean minimum blood oxygen desaturation of 67.7%. Continuous positive airway pressures between 5 and 15 cm of H_2O were again effective in eliminating apneic episodes. Sleep patterns were also restructured with stage III and IV sleep increasing from an average of 8.6% to 21.9% of the total sleep time. REM sleep also significantly increased from 10.2% to 30.3% of the total sleep time with NCPAP therapy. Desaturations per hour, measured as a greater than 2% drop from the baseline, averaged 77.7 without NCPAP therapy but only 8.2 while on the therapy. A six month check-up revealed 94% of the patients citing a subjective decrease in daytime sleepiness.

Current NCPAP Systems

Design

Current home-care systems available for the management of Sleep Apnea Syndrome rely on pressure produced from a continuously running centrifugal fan. Figure 3 is a schematic diagram of the current home therapy units. The operation of these units is quite simple; excess pressure and flow are relieved from the circuit by the spring loaded regulator present on the device. Standard 1-inch respiratory tubing is used to supply the pressure to the patient via a vinyl or silicone nasal mask. A non-rebreathing valve which provides for exhalation to the atmosphere against a set pressure is also present in the line.

The fans used in the current sleep apnea devices may provide anywhere from 20 to 120 liters per minute at pressure values up to 15 cm of H_2O. Higher pressure valves, up to 20 cm of H_2O, are available but these decrease the quantity of air flow. The most common pressure relief valve holds a constant airway pressure of 10 cm of H_2O. The device may be fitted to provide pressures at any one of the standard settings of 5, 7.5, 10, 12.5, or 15 cm of H_2O. The smallest standard pressure valve which still eliminates the apneic episodes is prescribed by the physician.

The operation of the regulator, shown in Fig. 4, is easily explained. The regulator is essentially two concentric cylinders with the inner cylinder having a spring-loaded circular cap, the blow-off valve, and input and
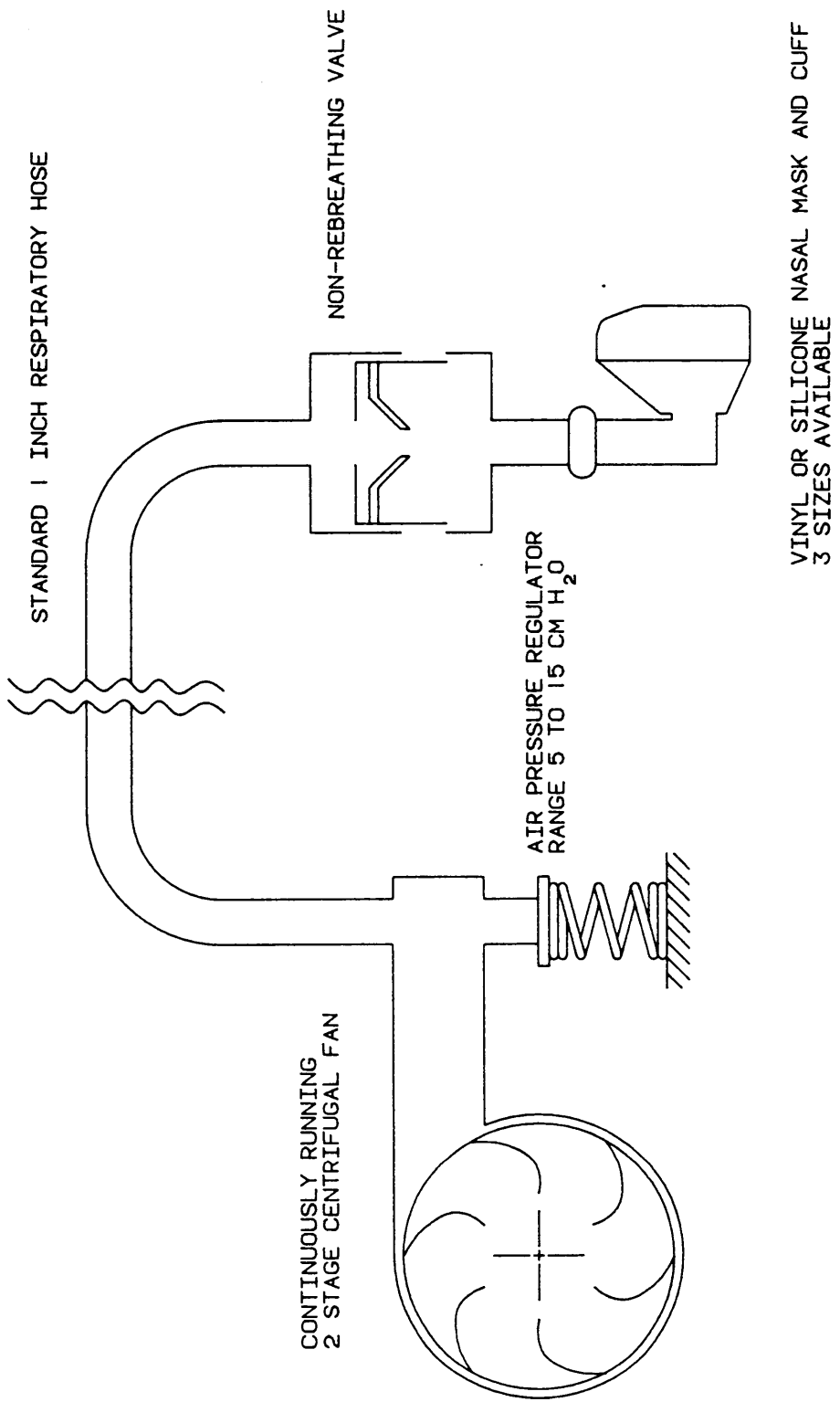
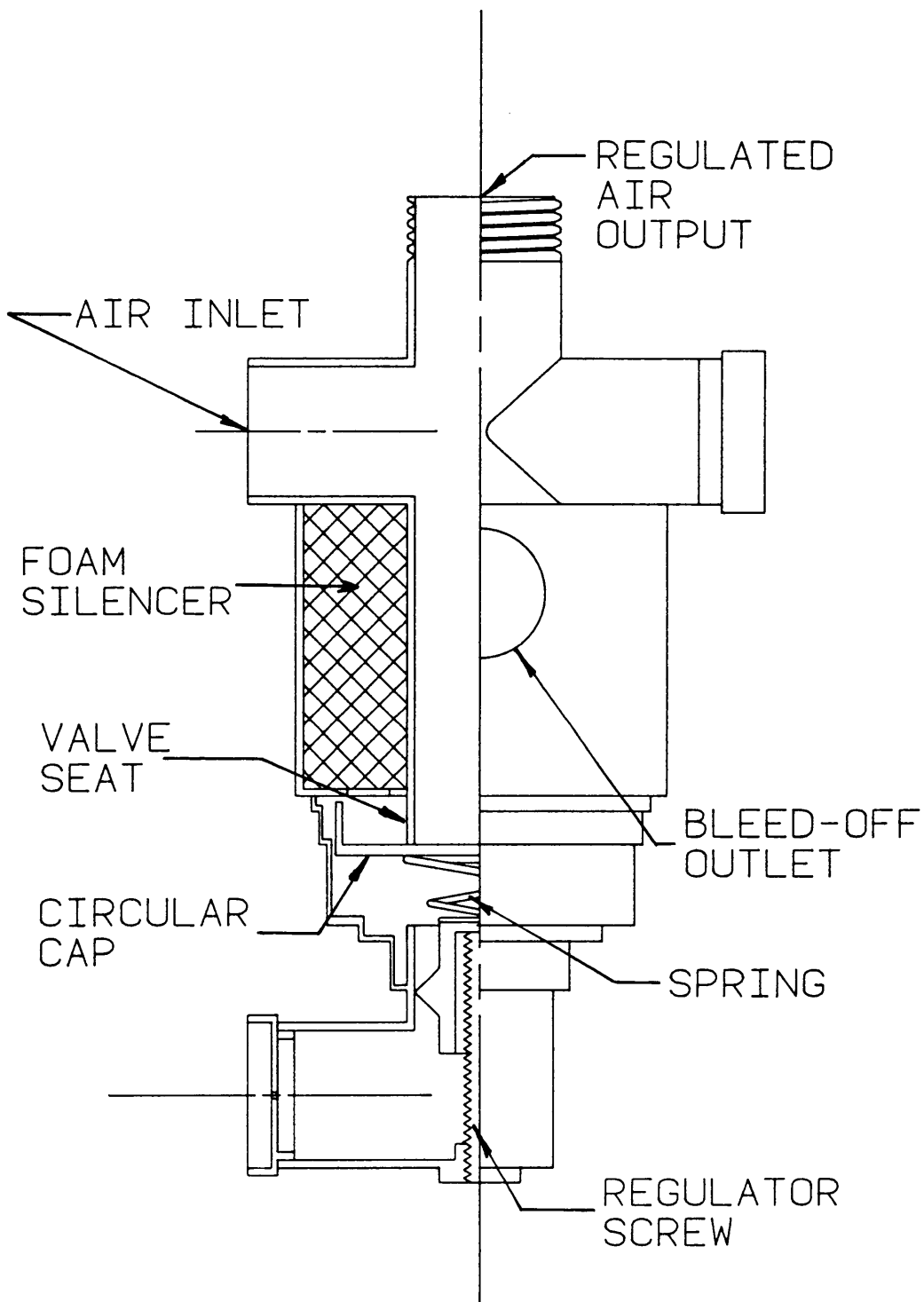FIGURE 3. SCHEMATIC DIAGRAM OF THE CURRENT FAN-TYPE NCPAP SYSTEM

FIGURE 4. CUT-AWAY DIAGRAM OF THE STANDARD REGULATOR
FOUND ON FAN-TYPE SYSTEMS

output lines. The outer cylinder houses a foam silencer which dissipates the noise of released air. Pressurized air enters the regulator through the air inlet and produces a force on the spring-loaded blow-off valve. When the desired pressure is exceeded, the force of the air pressure exceeds the force from the spring on the blow-off valve and the valve comes off its seat dumping the pressure to the atmosphere through the outer cylinder and bleed-off outlet. The preload on the spring, and thus the blow-off pressure of the regulator, is set by the length of the plastic regulator screw shown in Fig. 4. By allowing for the constant bleed of the system, the regulated air pressure supplied to the patient may be controlled adequately.

Operation of the non-rebreathing valve is similiarly easy to understand. Shown in Fig. 5, this cylindrical valve provides for leakproof passage of the pressure therapy and the pressurized discharge of exhalation gases. This is accomplished by the use of a scalloped sliding valve and Sierra® valve assembly within the non-rebreathing valve chamber. On inhalation, as shown in Fig. 5A, the sliding valve assembly is forced by the flow of air to the patient to cover the exhalation ports. Air flows through the sliding valve, around the displaced Sierra® valve, and to the patient. A silicone rubber collar permanently fastened between the sliding valve assembly and the housing limits the sliding valve assembly's travel to just cover the exhalation ports during patient inhalation. During exhalation, as shown in Fig. 5B, the pressure is raised on the patient side of the sliding valve assembly until the assembly begins to slide back against the force of the system's air pressure and partially reveal the exhalation ports. A balance between the forces of
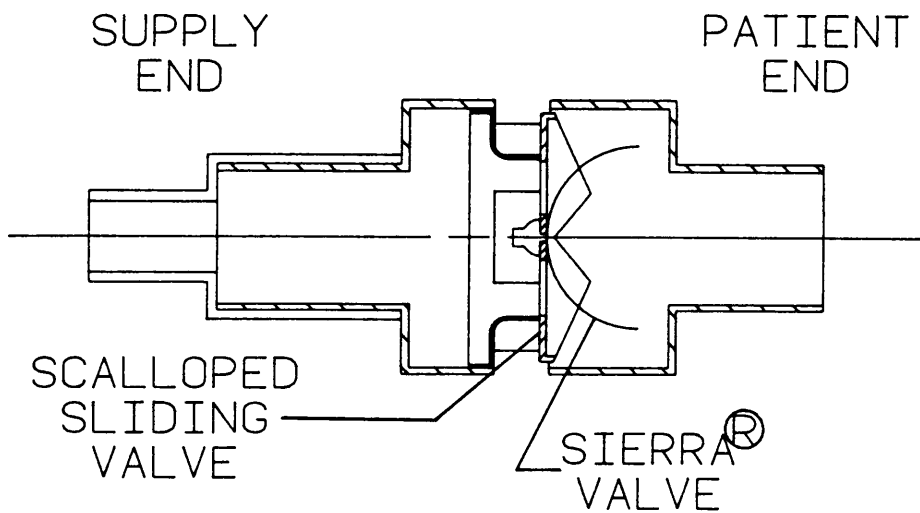
SUPPLY END PATIENT END

SCALLOPED SLIDING VALVE

SIERRA® VALVE

FIGURE 5A. STANDARD NON-REBREATHING VALVE DURING PATIENT INHALATION

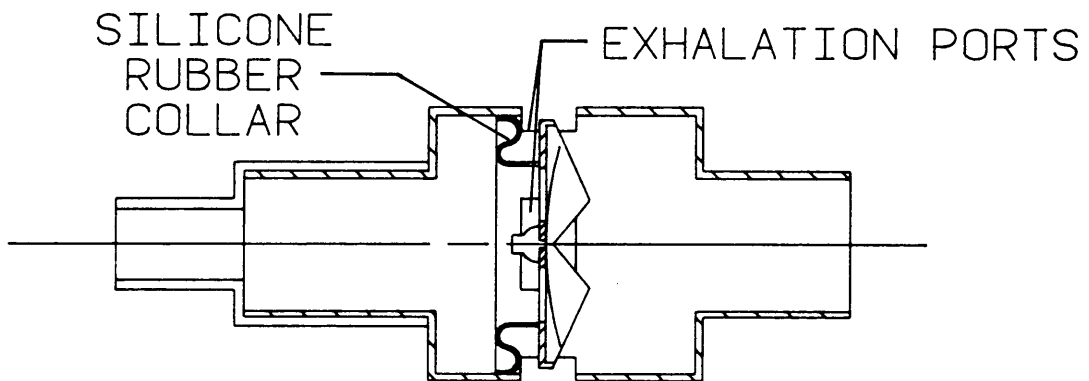SILICONE RUBBER COLLAR EXHALATION PORTS

FIGURE 5B. STANDARD NON-REBREATHING VALVE DURING PATIENT EXHALATION

the system's air pressure and the patient's exhalation pressure on the sliding valve assembly causes the sliding valve to remain in one position and allows for the pressurized exhalation of gases. Some system airflow may still pass around the Sierra® valve during patient exhalation; this wash-out removes more CO_2-rich exhalation gas. As the patient's exhalation pressure begins to diminish, the sliding valve assembly begins to slide forward and again occludes the exhalation ports. A leakproof passage is again provided and the process is repeated.

The vinyl or silicone nasal mask forms the critical patient/machine interface. A nasal mask is used instead of a full-face mask as a natural safety precaution. If a device malfunction should occur or the respiratory line should become clogged, the patient would automatically switch from nasal to oral breathing. In the worst case, the patient is returned back to his previous apneic breathing condition. In addition, the dead space volume of a nasal mask is less than that of a full face mask and will thus provide for better blood oxygenation and less CO_2 retention. If a mask's dead-space volume were comparable to the patient's tidal volume, little oxygen would be exchanged because of the re-breathing of exhalation gases. The hardshelled mask and cuff assembly is available in three sizes to allow for a good leakproof fit. The mask is held firmly in place over the patient's nose by two latitudinal Velcro® straps, one passing just below and the other passing just above the patient's ears. The cuff of the mask is held against the face by the pressure of the straps along with the pressure of the supplied air, providing a triangular line seal.

NCPAP home therapy has proven effective in the treatment of sleep apnea syndrome and it has been estimated that it is accepted by between 70% and 95% of those patients that show positive results to the therapy during an all-night sleep study [11,22]. The fan-type device used to deliver the required intratracheal pressures creates a stable pressure with little pressure fluctuation for different flows. The therapy provides a quick effective way to reduce daytime sleepiness and signs of cor pulmonale and hypertension, but may also produce unwanted side effects. Patients have complained of feelings of suffocation, nasal dryness, rhinitis, ear pain, and conjunctivitis as a direct result of using the NCPAP system.

One study stated that NCPAP patient compliance, as measured by use of the device for all but less than one hour per nightly sleep, appeared to be 75% [22]. To be included in the study, however, a patient must have shown satisfactory performance on a sleep study and also shown sufficient motivation to give NCPAP a try. Approximately 85% of those patients benefiting from the therapy were determined to possess sufficient motivation for trying NCPAP therapy. By requiring the study's participants to show a sufficient motivation, the authors are boosting the compliance statistics of the therapy. Keeping this and the patient's possible wariness in revealing non-compliance to his physician in mind, it may be more reasonable to assume the true compliance figure is in the range from 60% to 75%. This compliance rate lessens the appeal of current NCPAP systems.

Why would the patient compliance be so low on such an effective device? A compliance study of 24 recent SAS patients raised numerous complaints about current fan-type systems [22]. The 20 responding patients had been using the device for an average of 10.3 months, ranging from 1 month to 30 months, and showed varying use of the device. Sixteen patients revealed using the device for all nightly sleep, while an additional 2 revealed using the device for all but 1 and 2.5 hours of nightly sleep, respectively. Another respondent revealed using the device on alternate nights, while the final patient admitted that he did not use the device at all. Personal contact with the remaining four patients revealed that 3 of the 4 used the device sporatically while 1 used the device for all nightly sleep. Since there has been no indication that the therapy may be skipped without bringing back the apneic episodes, this compliance seems inadequate.

Most of the patients, 74%, complained of mask discomfort, specifically around the bridge of the nose. Air leaks at this juncture are thought to contribute to eye drying and subsequent conjunctivitis found in some patients. Oils secreted from the skin tend to harden the nasal mask cuff and create undue irritation of the skin surrounding the nose. The hardened cuff lessens the sealing ability of the mask and usually more head strap pressure is exerted to stop air leaks. Further increases in head strap pressure reduces blood flow to the skin and pressure necrosis results. The second most common complaint, nasal dryness and congestion, was cited by 68% as a major problem with the therapy. The lack of provisions for air humidification in current machines is cited as the reason for increased nasal dryness

and irritation. Many patients have such a great problem that they must employ drug or spray therapy to treat their nasal ailments. Other objections cited in this study included 5 noise complaints, 2 cases of morning rhinorrhea, 2 cases of chest discomfort, and 1 case of intra-nasal abrasions.

Noise complaints are common, with some patients blaming this disturbance as the main reason for not sleeping with their spouses. Recent development work in fan geometry and port configuration by Respironics has reduced the noise level of of their most current device, though. Noise disturbance, the major impetus for the positive-displacement pump, has been essentially eliminated. A 38-patient study found that only 36% of the patients considered the device comfortable [26]. It is hoped that by employing a custom fit nasal mask with an inproved exhalation valve design causing less CO_2 retention on a quiet system that supplys humidified air, the patient compliance and hence therapy effectiveness will rise dramatically.

Positive-Displacement Pump

The treatment of Sleep Apnea Syndrome with the new positive-displacement pump still relies on the effectiveness of NCPAP therapy. The main difference between present NCPAP therapy systems and the system proposed here is in the generation of the pressurized air. Present systems rely on a continuously running fan and a pressure regulator to provide the prescribed level of NCPAP therapy while the new system employs an electronically-controlled motor-driven positive-displacement bellows pump for pressure generation and control. This concept is proven feasible because it can be shown that the performance of the positive-displacement pump is comparable to the current fan-type NCPAP systems. There are 5 main components to the new device: (1) the plastic bellows donated to Dr. Arp which act as the air supplier, (2) the 115 vdc motor which drives the linkage to compress and expand the bellows, (3) the pressure sensor and related control circuitry which supplys the motor with the hybrid digital/analog control commands, (4) the welded polyethylene reservoir which acts as a capacitor for the smoothing and storage of pressurized air, and (5) the custom nasal mask and exhalation valve assembly which forms the important patient/machine interface and allows for the pressurized exhalation of gasses.

The schematic diagram of Fig. 6 shows how these elements come together to form a NCPAP system. The electric motor drives the bellows with a simple slider crank mechanism. Pressurized air, developed by the motion of the bellows, travels through a single Tygon® tubing supply line
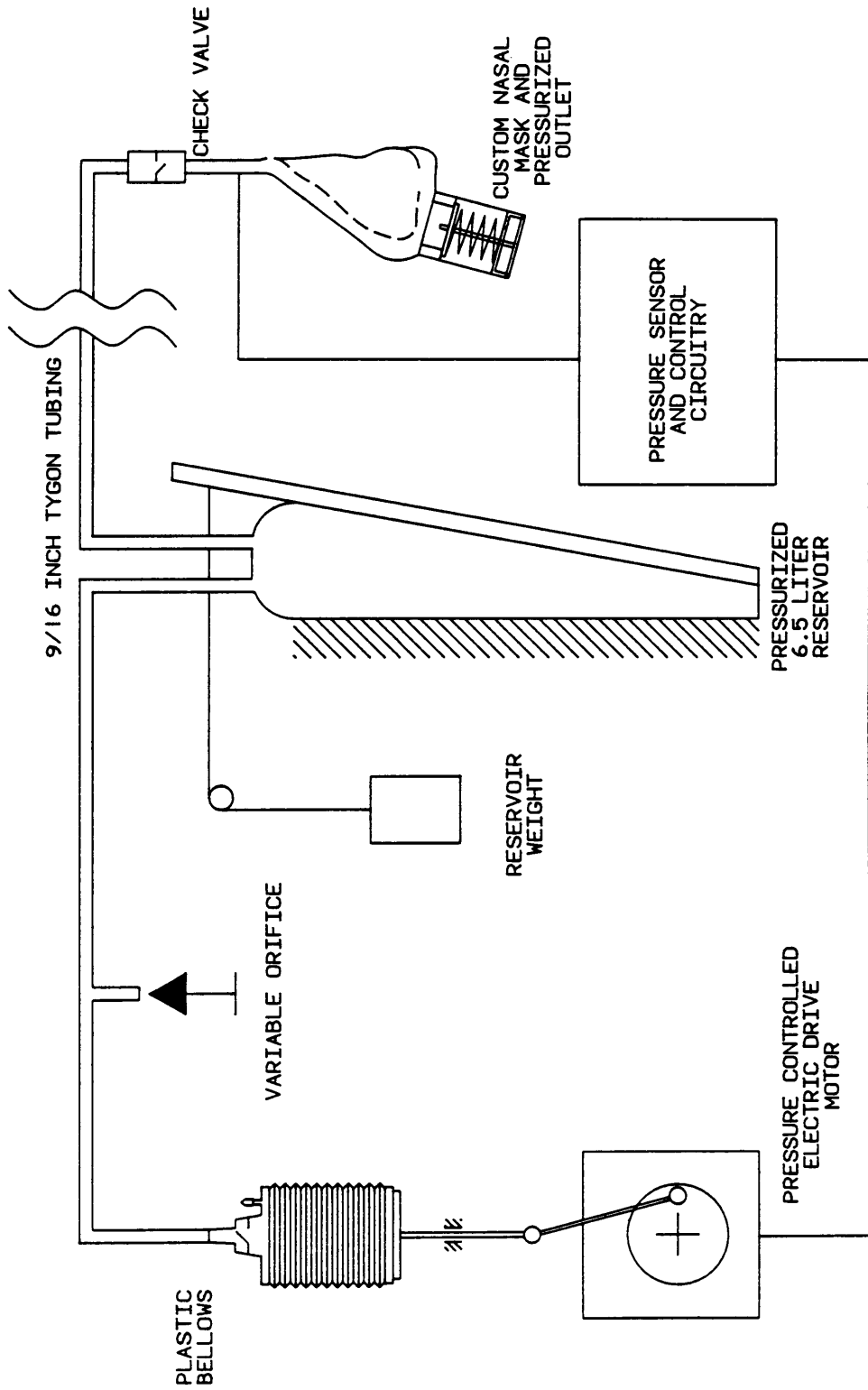
Figure 6. Schematic diagram of the positive displacement NCPAP system

past an orifice. The orifice provides a flow route for air during patient exhalation allowing for a higher idling motor speed during patient exhalation. The pressurized air is fed into a 6.5 liter reservoir. As the reservoir fills, a weight is lifted which keeps pressure on the reservoir and provides a limit on the reservoir volume. A spring-loaded disk valve located on the patient's custom nasal mask provides for the exhalation of gasses against the prescribed air pressure. A check valve located just above the nasal mask insures that no rebreathing of exhalation gasses is possible. The custom nasal mask is molded from acrylic to fit the individual patient and has the exhalation valve built in. A pneumatic sensor monitors the air pressure at the nasal mask and produces a proportional analog signal for the motor controller circuitry. This analog signal is used to control the motor to provide a stable nasal pressure.

Each of these components of the positive-displacement NCPAP system will be discussed in detail in the following sections. Detailed dimensioned drawings, where not available within the text, are included for reference within Appendix B found at the end of this thesis. Since the mechanical engineering machine shop where the device was built uses the English system of units, the dimensions of the machine parts are given in inches. A table of metric equivalents, giving the English dimensions in inches and SI dimensions in millimeters, is also included in Appendix B.

Plastic Bellows

The plastic bellows used in the new NCPAP system are modified versions of the commercially available Breath Saver® resuscitator designed for a single hand-actuated use. The bellows, shown in the cut-away schematic of Fig. 7, comes complete with integral rubber output and input check valves to seal the chamber during air input and output, respectively. The existing bellows were modified to create a point of attachment for the push rod drive mechanism through a 1/4 - 20 tapped plexiglass mounting disk. The vibration of the output valve's rubber tongue was eliminated by providing a plastic support to keep it from aligning with the flow. The plastic bellows has an unvalved port through which humidification of the pressurized air may be introduced. Humidification of the air, a major attribute to a successful NCPAP system, is possible with this prototype device. The mechanism for providing the humidification is a device designed and built by Dr. Arp of the Bio-medical Engineering Laboratory. Its operation will not be discussed in this thesis; however, it is noted that the positive-displacement prototype device has a provision for selectable, controlled humidification of the inhaled air.

The operation of the bellows is quite simple. When the driving push rod is extended and the bellows fully compressed, the output check valve closes after delivering a charge of air. Upon expansion of the bellows, air is drawn into the bellows through the milled channels in the mounting disk and through the input valve. Compression of the bellows causes the
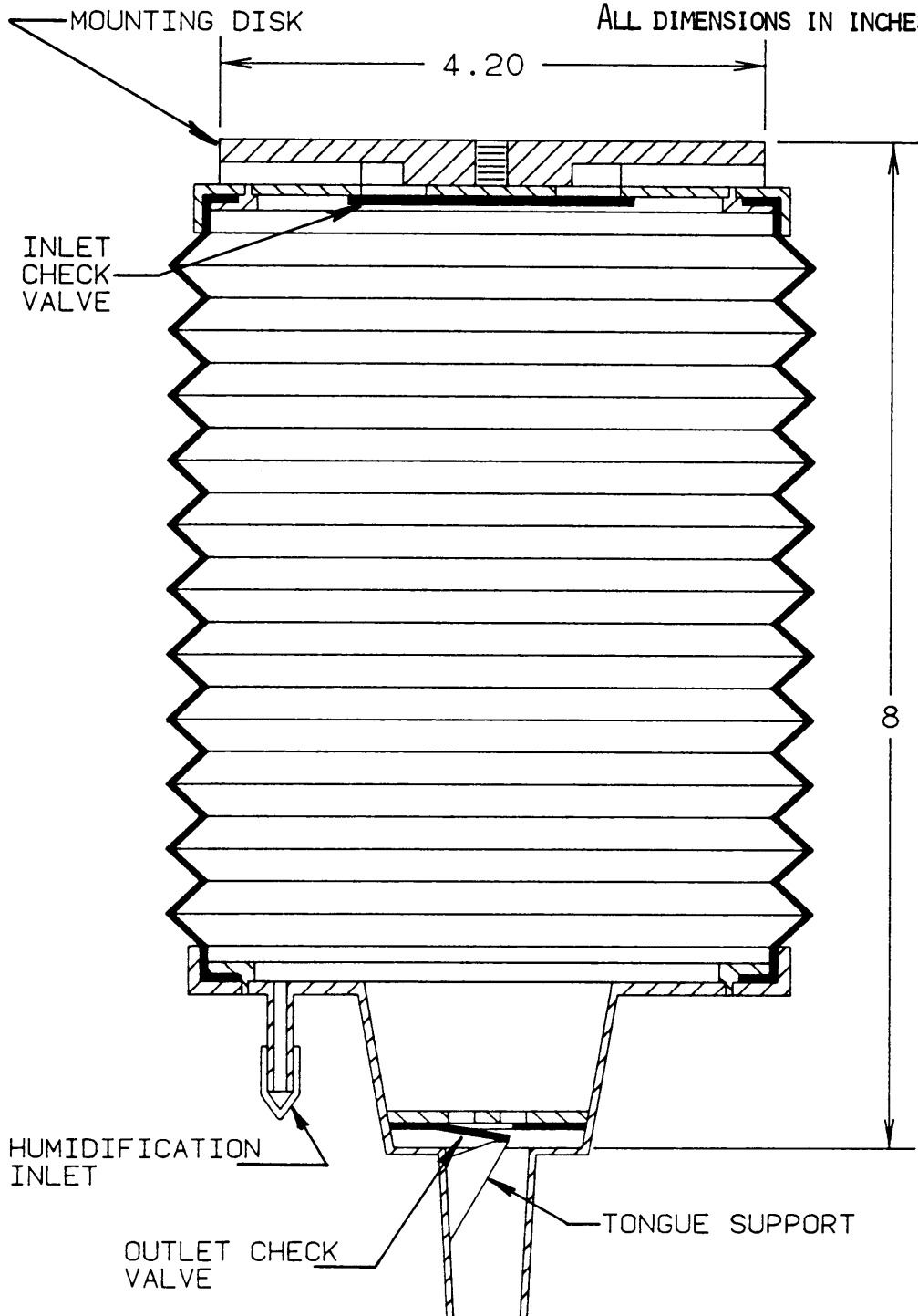
FIGURE 7. CUT-AWAY DIAGRAM OF THE MODIFIED BELLOWS

input valve to seat and the output valve to open. Air is then driven from the bellows into a pressurized supply line. As the bellows again expand the output valve is closed by the higher supply line pressures and the process is repeated. Four plastic bellows are employed in the NCPAP device and their compressions are phased at 90 degree intervals. When the first bellows is at its most compressed state, the second bellows is at its most expanded state. The two remaining bellows are in a partially collapsed state with one expanding and the other compressing. A tapered adapter holds the output end of the bellows in place and provides the connection to the pressurized air line through a standard barbed plastic fitting.

Electric Motor Drive

The bellows are driven as the slider in a slider crank mechanism by a 115 vdc Bodine electric motor. This motor, with a speed-reducing gearhead, was used in the prototype device because of availablility and its ability to provide adequate power in order to drive the linkage. The motor provides a rated power of approximately 1/80 horsepower at a rotational speed of 20 revolutions per minute and 40 inch-lbs of torque. The motor configuration and its over-all dimensions are shown in Fig. 8. Because the stated speed of the motor was deemed insufficient to meet the transient response characteristics of the NCPAP system, an external gearset with a speed increase of 4 to 1 was employed. Figure 9 gives the mechanical details of the drive gearset. The motor drives the 128-tooth, 24-pitch spur gear directly through its 12.7 mm (0.5 in.) hub. Geared to this through the
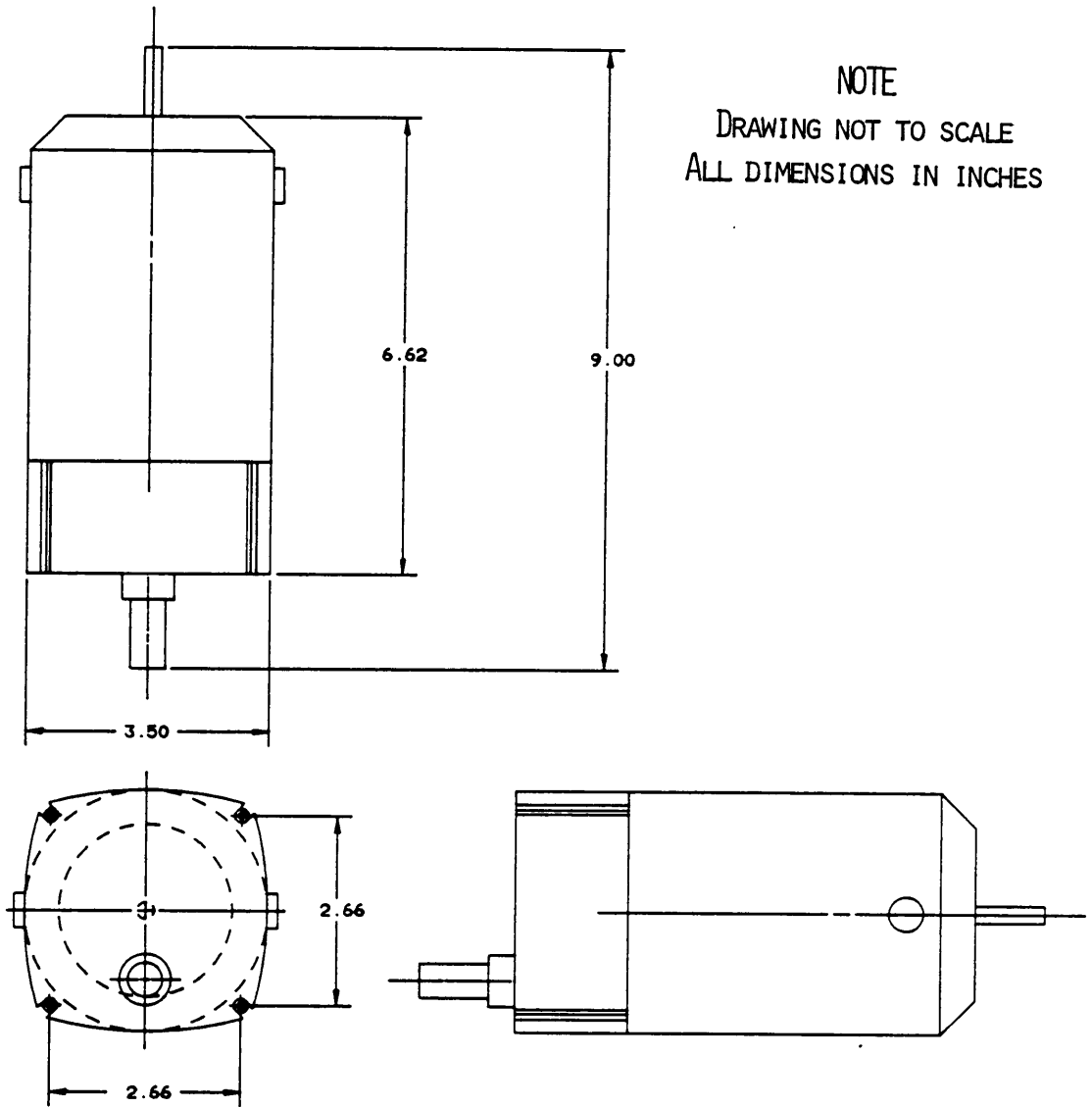
NOTE
DRAWING NOT TO SCALE
ALL DIMENSIONS IN INCHES

6.62

9.00

3.50

2.66

2.66

FIGURE 8. BODINE MOTOR CONFIGURATION AND OVERALL DIMENSIONS
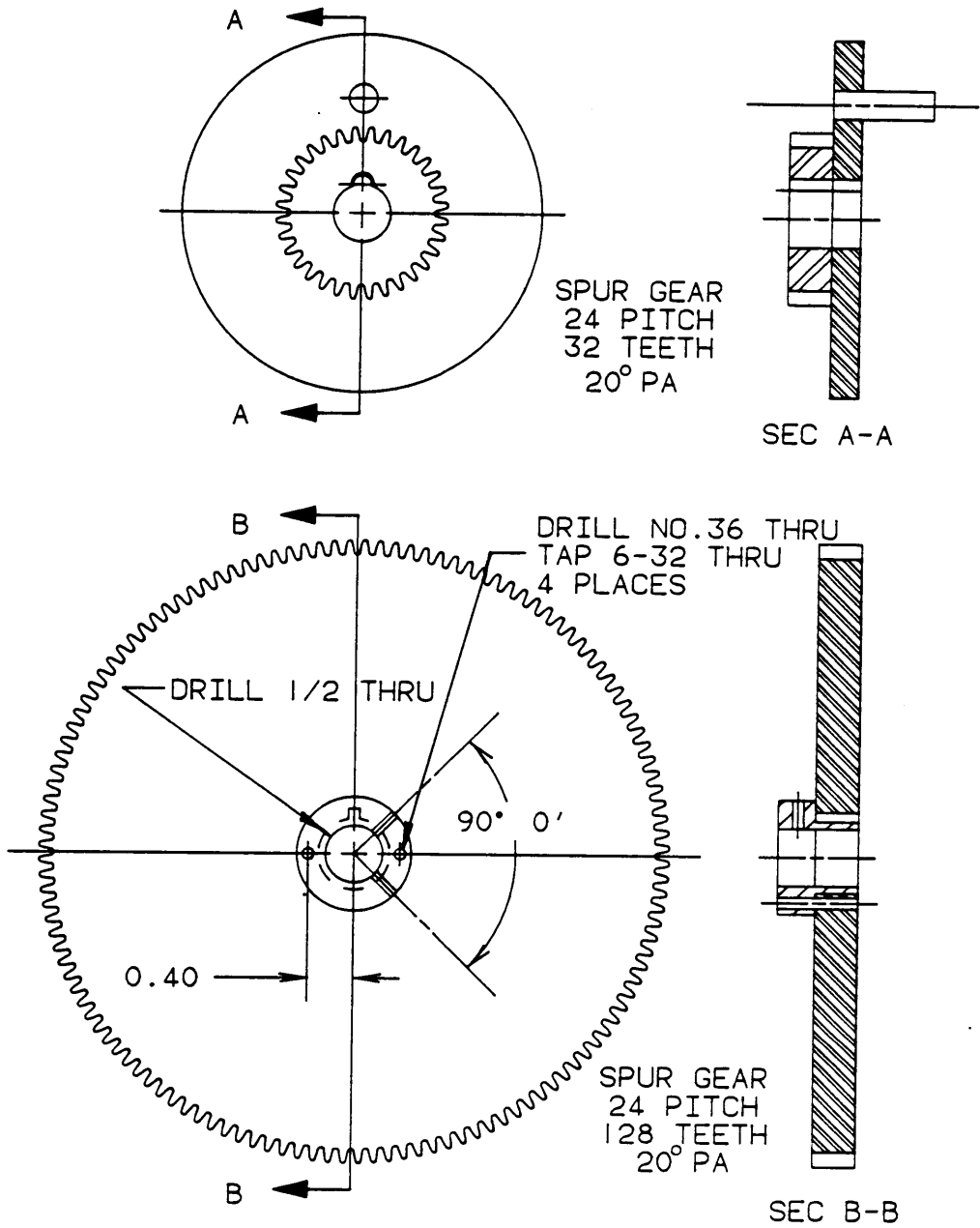
NOTE
DRAWING NOT TO SCALE
ALL DIMENSIONS IN INCHES

A

A

SPUR GEAR
24 PITCH
32 TEETH
20° PA

SEC A-A

B

DRILL NO.36 THRU
TAP 6-32 THRU
4 PLACES

DRILL 1/2 THRU

90° 0'

0.40

SPUR GEAR
24 PITCH
128 TEETH
20° PA

B

SEC B-B

FIGURE 9. PROPOSED IICPAP SYSTEM'S GEARING

32-tooth, 24-pitch spur gears are two sets of crankshafts used to drive the bellows. Because of this gearset, the bellows may be driven at a maximum frequency of 80 cycles per minute. The crank journal on the crank shaft is offset from the rotational centerline by 25.4 mm (1.0 in.) so that the total crank throw is 50.8 mm (2.0 in.). With each revolution at full motor speed each bellows can supply approximately 400 ml of air, nearly 32 liters per minute.

Pressure Sensor

The pressure sensor and associated control circuitry is the heart of the positive-displacement NCPAP device. Without a regulator to limit the supplied air pressure, the controller must limit the motor speed in order to produce air at the prescribed air pressure level for varying patient flow rates. The sensor, shown in the cutaway of Fig. 10, is fabricated from a brass diaphragm pressure gauge. The kinematic linkage of the pressure gauge is removed and replaced with a GE H21A1 phototransistor and a mylar flag. The mylar flag is made from a photographic negative which has been developed with half the flag opaque. Pressure input to the brass diaphragm causes the diaphragm to deflect and the flag to rise. When the flag rises enough to begin blocking the light path between the phototransistor's light-emitting diode (LED) and the photo-detector, the transistor's collector junction voltage begins to rise. The voltage swing of the transistor with a +5 v supply ranges from 0.7 v with a transparent flag and no blockage of light to +5 v when the flag is blocking all the light. The
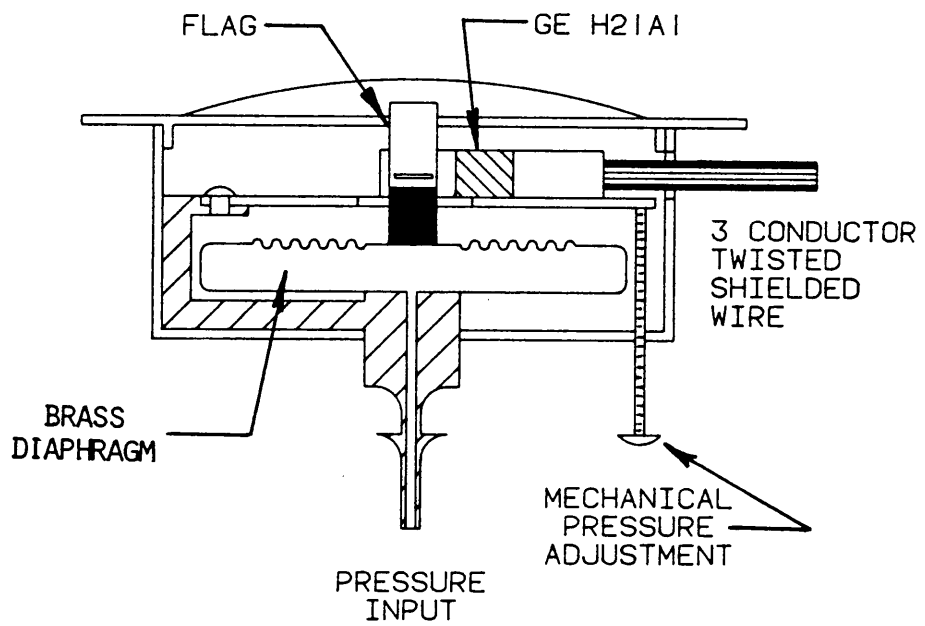
FIGURE 10. CUT-AWAY DIAGRAM OF THE PRESSURE SENSOR

phototransistor package is mounted on a thin cantilevered aluminum beam to allow for small adjustments in pressure setting. These can be made by turning the mechanical pressure adjustment screw on the sensor. A second fine pressure adjustment is available on the main control circuit board. Three wires are returned to the main control circuit board from the sensor via twisted shielded wire to limit any induced noise on the pressure signal. The wires return the transistor's collector junction, the light-emitting diode anode, and ground potential to the main control board.

Control Circuitry

The Bodine electric motor is controlled with a simple circuit that relies on the pressure signal returned from the pressure sensor. This circuit acts as a pulse-width modulator creating control pulses of varying width in proportion to the pressure signal voltage. This pulse-width modulator's output is fed into a proprietary motor control circuit developed by Dr. Arp for final control of the motor. The controller's schematic circuit is shown in Fig. 11. The functional electrical element of the pressure sensor, the GE H21A1 phototransistor is also shown on this schematic. The operation of the circuit is based primarily on the operation of a single LM324 operational amplifier chip. This integrated circuit chip contains four low cost, reliable operational amplifiers labelled 324A, 324B, 324C, and 324D in the circuit diagram. Amplifiers 324C and 324D are wired as a standard triangular wave oscillator. By varying the capacitance or the 324C amplifier's feedback resistor value, one may easily change the period of oscillation. In the
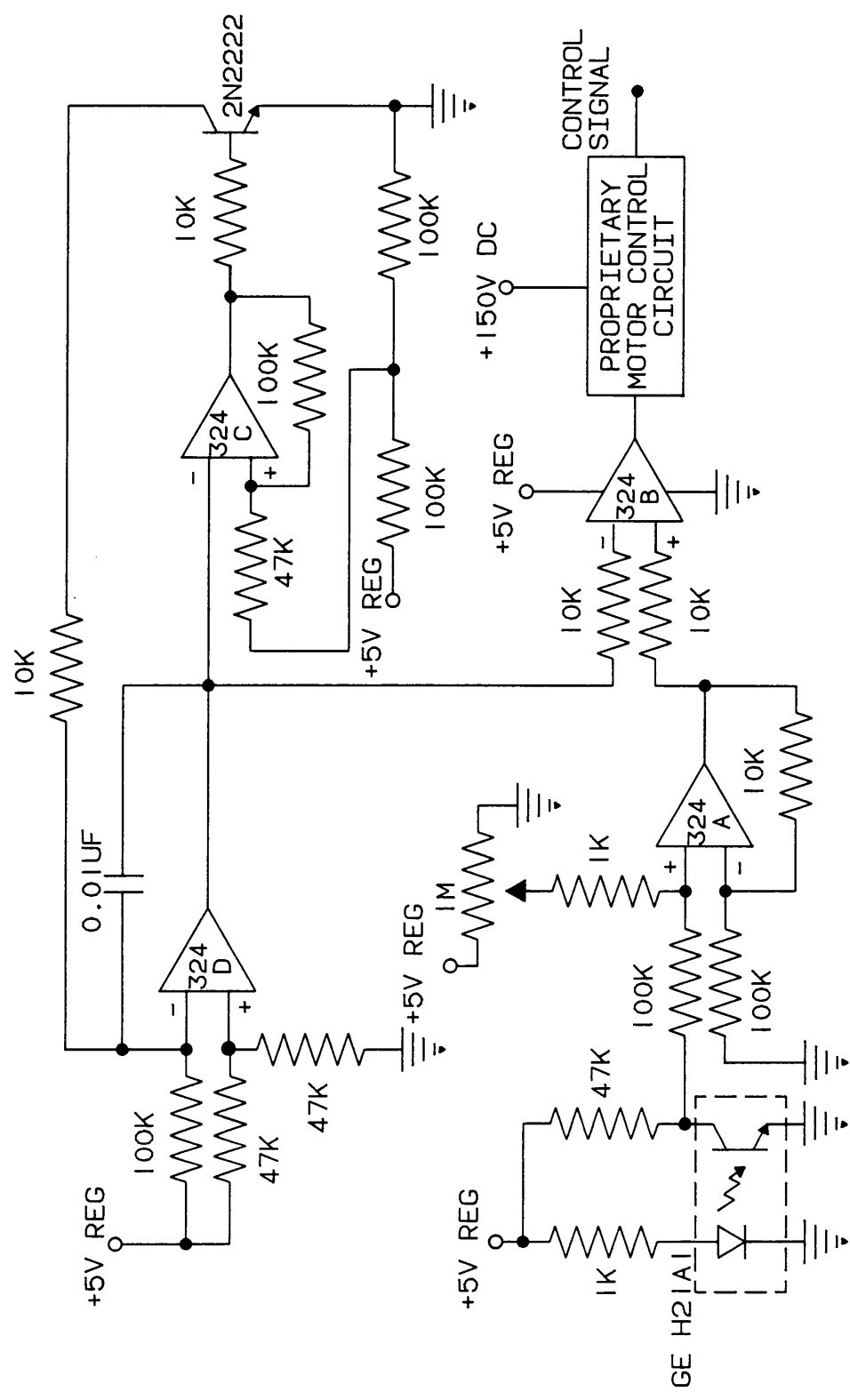
Figure 11. Electrical schematic diagram of the motor controller circuit

configuration shown in Fig. 11 the triangular wave oscillator has a period of 1.1 ms. The triangular wave has a rise-time of 0.1 ms and a decay-time for the remaining 1.0 ms.

The phototransistor output, ranging in voltage from +0.7 vdc to +5 vdc, is added to the voltage picked off the 1 M ohm trim potentiometer that makes up the fine pressure adjustment mentioned earler. After amplification by the 324A operational amplifier, this voltage is compared to the output of the triangular wave oscillator by the 324B operational amplifier acting as a comparator. When the phototransistor voltage is low, indicating a low pressure, the comparator creates short logic level 1 pulses with long logic level 0 spaces between them. As the pressure rises, the phototransistor output rises and the logic level 1 pulses get proportionately longer while the logic level 0 spaces get proportionately shorter. This pulse-width modulated signal is used by the proprietary motor-speed control circuit for the Bodine motor.

The +5 vdc and +155 vdc sources necessary to run the logic elements of the motor controller and alarm system are generated by the power supply circuit of Fig. 12. This schematic shows the generation of a stable regulated +5 vdc supply as well as an unregulated but filtered +155 v peak supply. Line voltage, 115 vrms, is brought into the device and isolated from the device's circuitry with a 1-to-1 isolation transformer. This 115 vrms voltage is immediately rectified with a full-bridge rectifier and filtered with a 1000 uf capacitor for a stable +155 vdc source. The +5v source is produced by
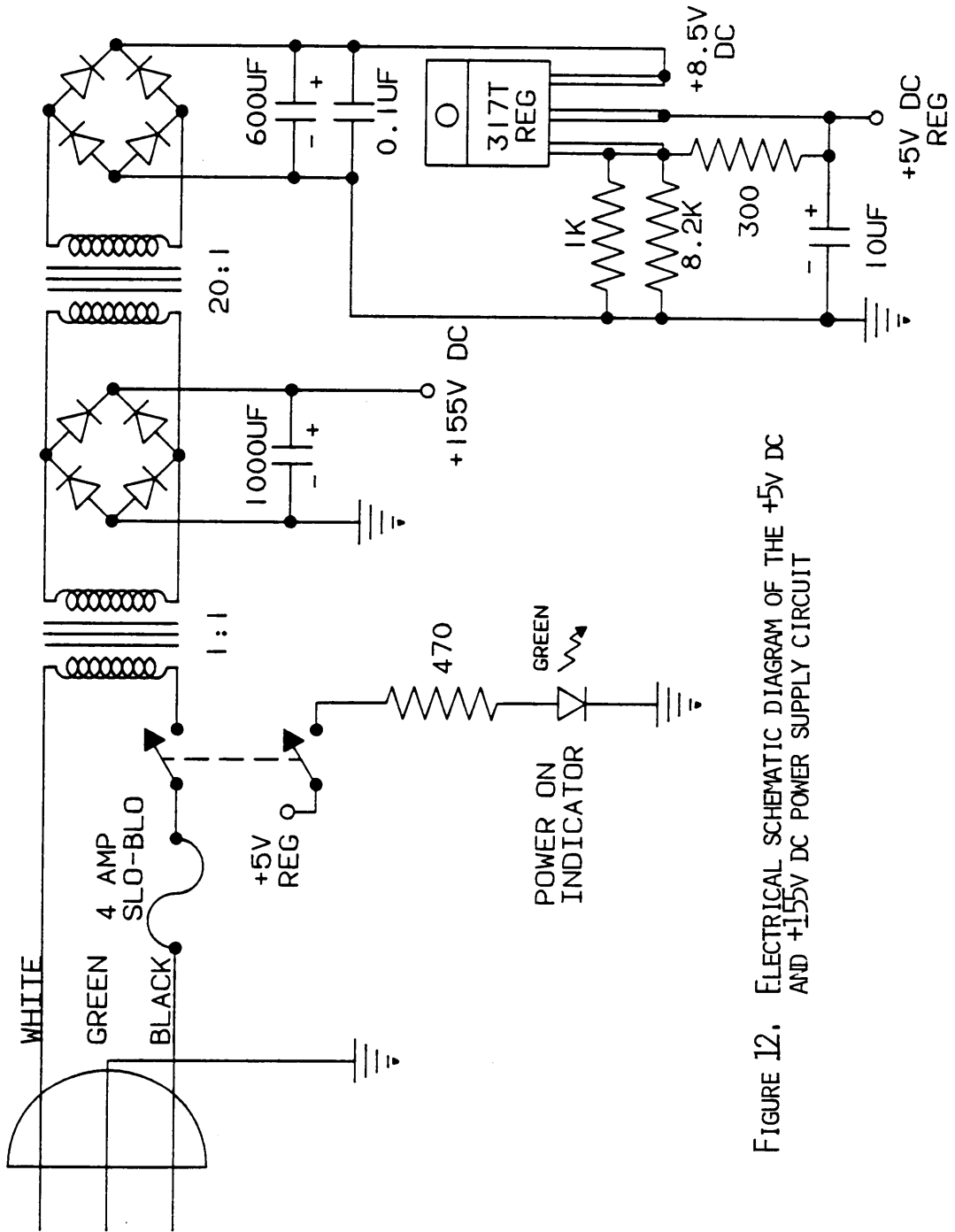
FIGURE 12. ELECTRICAL SCHEMATIC DIAGRAM OF THE +5V DC AND +155V DC POWER SUPPLY CIRCUIT

the transformation of 115 vrms to 6 vrms through a 20-to-1 transformer. This 6 vrms voltage is then passed through a full bridge rectifier and filtered with a 600 uf capacitor. This creates an unregulated 8.5 vdc supply. This voltage is input to a LM317T variable voltage regulator for effective generation of a highly stable regulated +5 vdc supply.

Pressure Storage and Filtering

Just as the capacitors found on the power supply circuit of Fig. 12 are used to smooth the voltage fluctuation created after rectification of an alternating current source, the weighted pneumatic reservoir smooths the pressure fluctuation caused by the pulsed generation of pressure from 4 out-of-phase bellows. The welded polyethylene reservoir, Fig. 13, is kept under pressure by a 3 pound suspended weight. As the pressure in the supply line begins to increase, the reservoir volume tends to increase and reduce the pressure fluctuation on the patient supply line. Similiarly, as the patient supply line pressure begins to decrease, the weight squeezes the reservoir together which lessens the reservoir's volume and reduces the pressure drop in the patient supply line.

The weighted reservoir also increases the total volume of pressurized air available for those patients requiring large inhalation volumes which may exceed the device's capacity to produce instantly. The 6.5 liter reservoir has a single inlet into which the pressurized air of all four bellows enter and a single outlet from which the patient is supplied pressurized air. The

NOTE
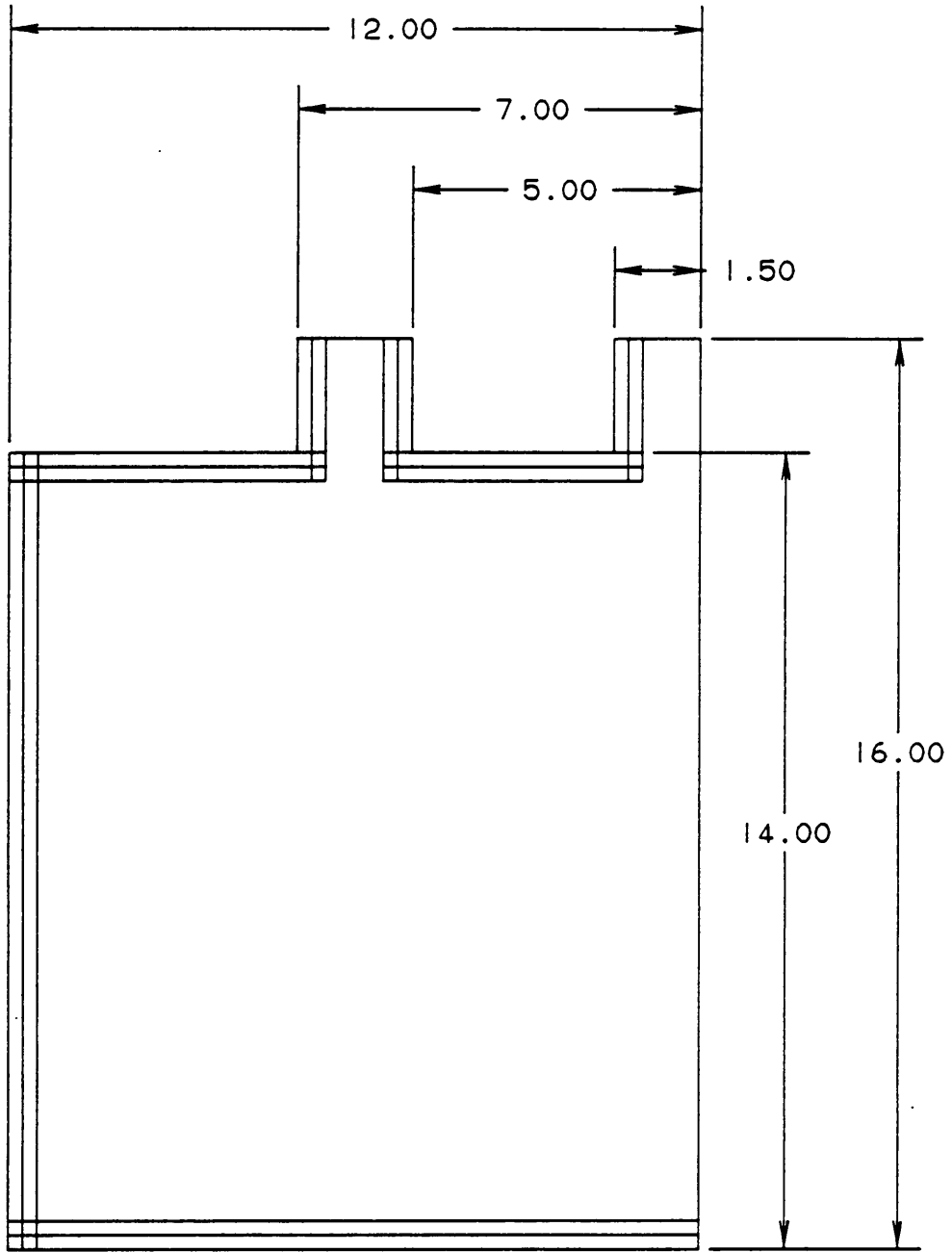DRAWING NOT TO SCALE
ALL DIMENSIONS IN INCHES

FIGURE 13. 6.5 LITER POLYETHYLENE RESERVOIR

operating volume of the capacitor can be changed by replacing the suspended weight with another weight of different mass or by changing the setting of the variable orifice found in the patient supply line. A small orifice causes the reservoir pressure to increase and thus produces a larger reservoir volume. Similiarly, a larger orifice will cause the reservoir pressure to drop and produce a correspondingly smaller reservoir volume. In this way, the orifice may be used to fine tune the pressure level of air supplied to the patient.

Custom Nasal Mask and Exhalation Valve

Uncomfortable and ineffective nasal masks found on present NCPAP devices have prompted the development of a comfortable and reliable custom nasal mask and exhalation valve combination. Because the nasal mask used on the positive-displacement NCPAP system is fitted for each individual patient, it offers a distinct comfort advantage over the present mask. The mask is sealed more effectively because it relies on a large sealing area rather line-type seal. Furthermore, since present mask and valve combinations have a dead space of approximately 110 ml which is over 25% of the tidal volume, combining the nasal mask and exhalation valve into one unit essentially eliminates the dead-space volume problem. With the new mask and valve combination there is virtually no dead space volume for rebreathing of exhalation gas. This should provide for a huge reduction in CO_2 retention by the patient.

The custom fit of the nasal mask is produced by replicating the form of a patient's nose in plaster of paris. To do this, a negative image of the nose is first made in wax. A cup of warm wax is gently molded over the patient's nose while the nares are held open with cotton. The wax is allowed to cool and stiffen before a plaster of paris positive image of the nose is produced. The new mask and valve assembly, shown schematically in Fig. 14, is created by laying a thin film of Lang Dental Acrylic over the plaster of paris form of the patient's nose. A bead of wax approximately 12.7 mm (0.5 in.) in diameter is then laid down the ridge of the nose to the opening of the nares. This will form the air passage once the mask is complete. A standard barbed fitting is inbedded in the wax at the bridge of the nose to serve as the mask's air supply fitting. With the valve seat in place at the tip of the nose near the external nares, a second layer of dental acrylic is laid over the form. To finish the mask, the wax is removed and the internal openings for the nares produced. A check valve that acts as a safety valve to insure that the CO_2-rich exhaled gasses leave the mask and are not rebreathed, may be incorporated within the mask as well. Presently, it is a seperate valve located just prior to the inlet of the custom nasal mask and exhalation valve assembly.

The exhalation valve assembly, shown in the cut-away drawing of Fig. 15, allows for the exhalation of gasses against a set pressure and also permits the purging of the air supply line. By allowing for a small biasing flow of air through the exhalation valve assembly at all times, the mask retains little if any CO_2. Current NCPAP sytems require the exhalation of gas through
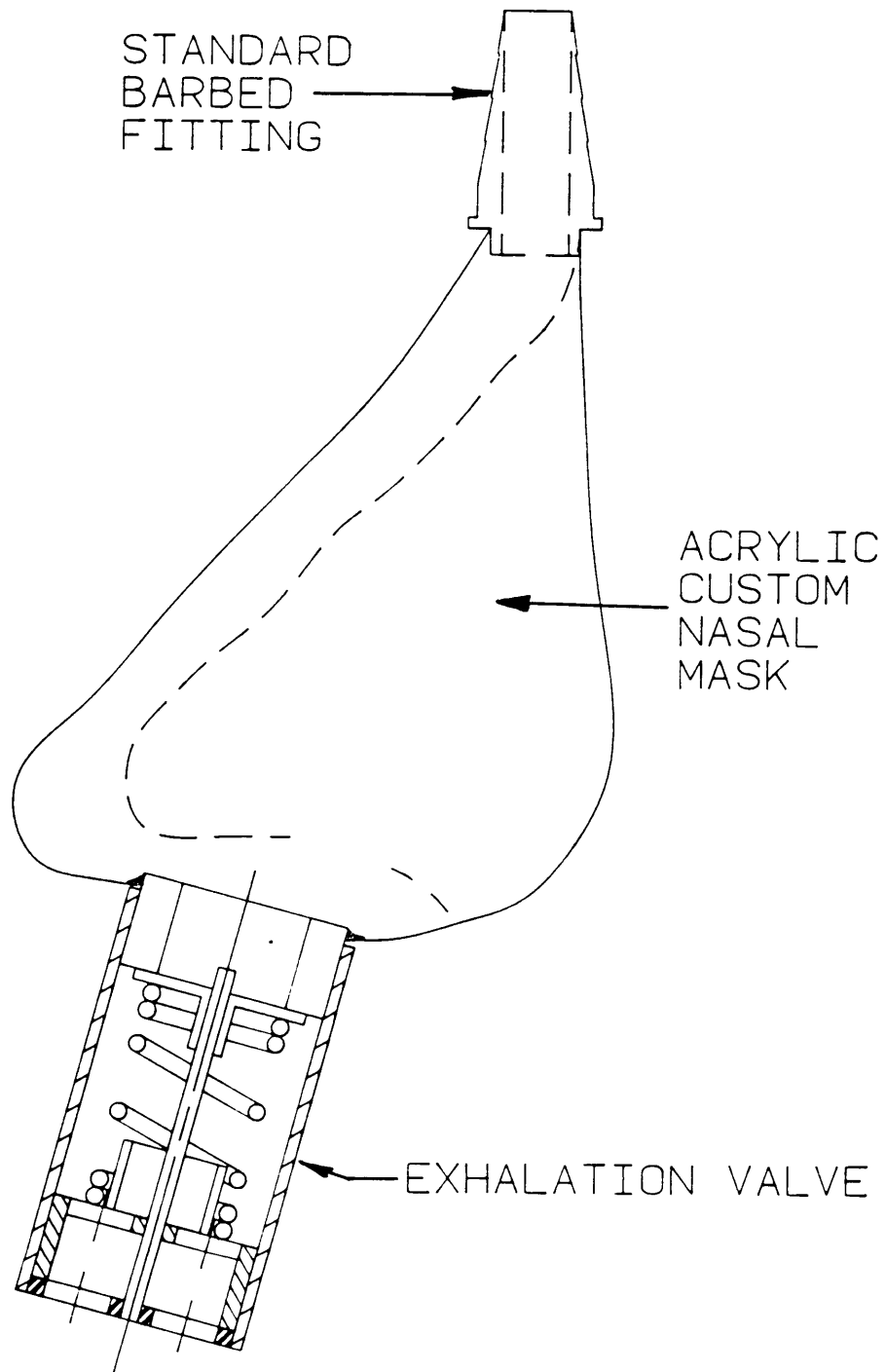
STANDARD
BARBED
FITTING

ACRYLIC
CUSTOM
NASAL
MASK

EXHALATION VALVE

FIGURE 14. CUT-AWAY DIAGRAM OF THE CUSTOM NASAL MASK AND EXHALATION
VALVE

NASAL MASK

VALVE
SEAT

DISK
VALVE

VALVE
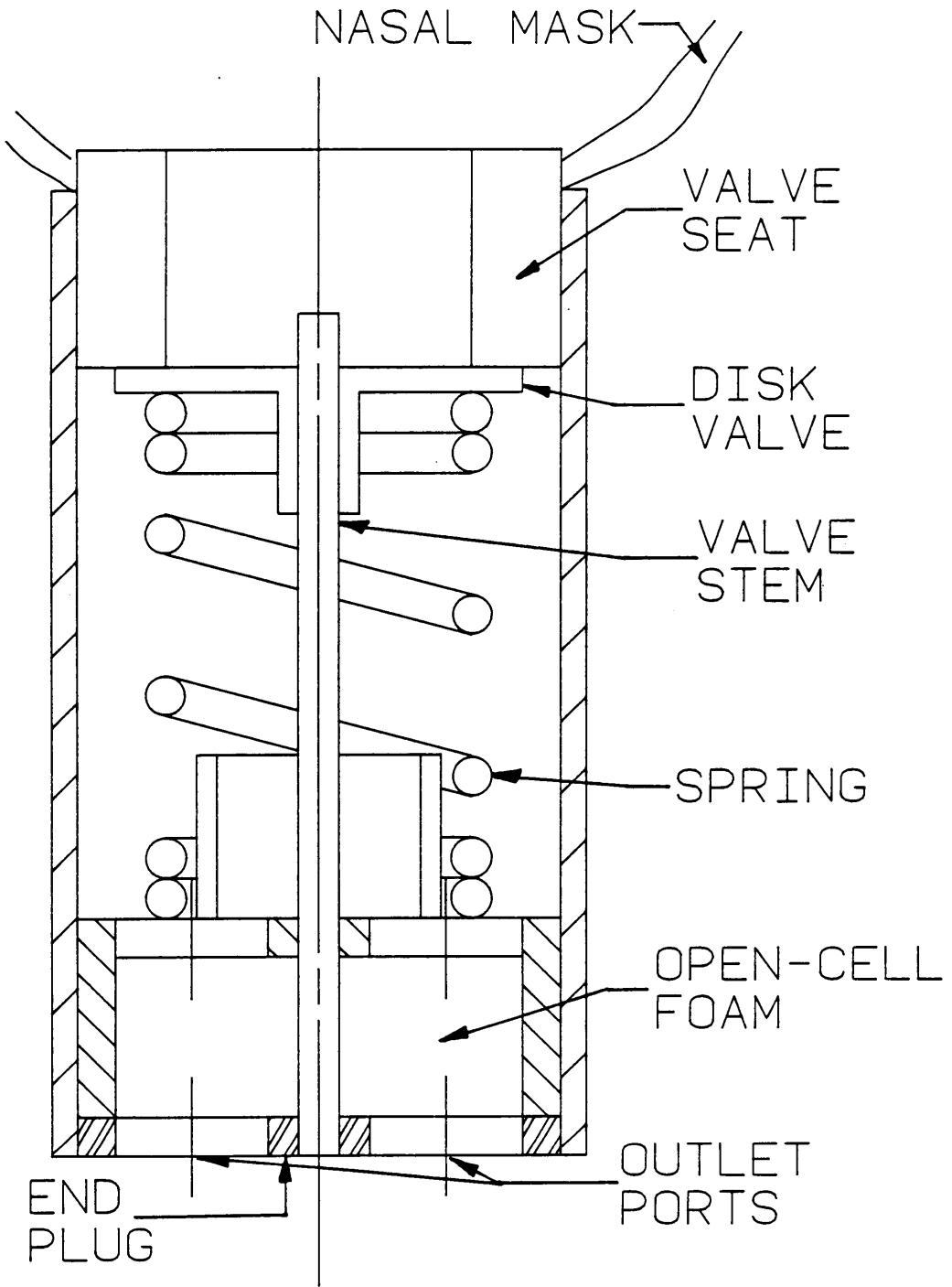STEM

SPRING

OPEN-CELL
FOAM

END
PLUG

OUTLET
PORTS

FIGURE 15. CUT-AWAY DIAGRAM OF THE EXHALATION VALVE ASSEMBLY

the same hose that supplies fresh air. The volume of the mask and valve assembly thus becomes added dead space to the patient's respiratory system. With the new mask this dead space is virtually eliminated. The principle airflow during inhalation is to the patient, however the spring is sized to allow the disk valve to just break loose of the valve seat and allow a small purging airflow. On exhalation the spring-loaded disk valve holds the prescribed pressure while exhaust gases are released. A small layer of open-cell foam within the valve reduces the noise of exhalation and the purging air flow.

Low-Pressure Monitor and Alarm

A unique feature to the positive-displacement NCPAP device is the low-pressure monitor and alarm. This system, shown schematically in Fig. 16, monitors the pressure at the patient/machine interface and if it does not meet a minimum threshold within 10 seconds triggers a visible red LED alarm. In addition to the visible alarm, a 2 kHz pulsating tone is activated when the patient-selectable alarm defeat switch is in the "enable" position, . A 33 second start-up disable has been incorporated within the device to allow an alarm-free period for the patient to make mask adjustments. During this time the activation of the alarm is automatically blocked. If the alarm defeat switch found on the control panel is in the "defeat" position, the audible tone will not be activated; the visible red LED cannot be defeated, however. Another red LED warns the patient when the "defeat" position has been selected.
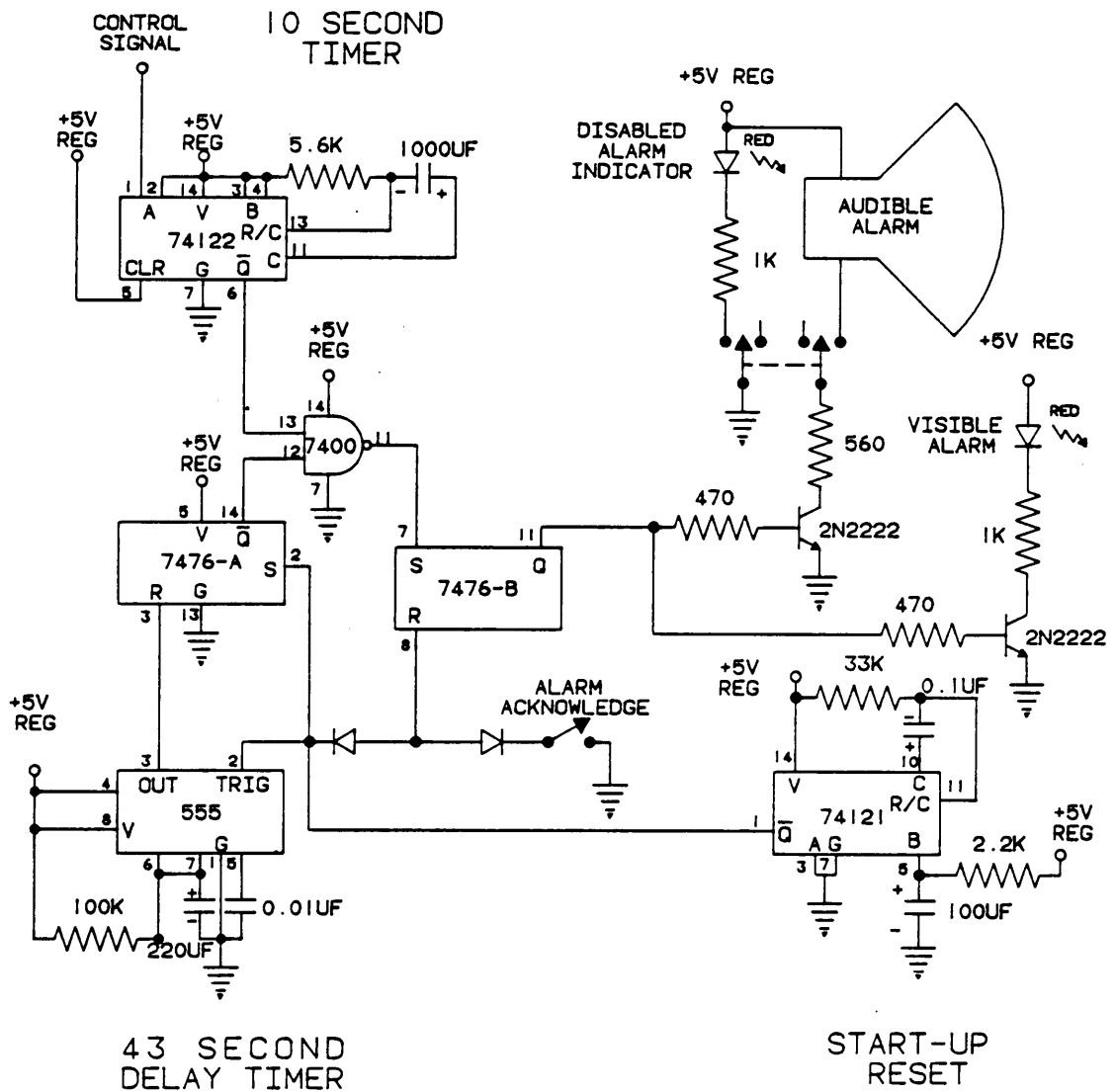
FIGURE 16. ELECTRICAL SCHEMATIC DIAGRAM OF THE LOW PRESSURE
ALARM CIRCUIT

If the alarm should sound, it may be acknowledged by first replacing the nasal mask, if it has fallen off, and then pressing the alarm acknowledge button on the control panel, shown in Fig. 17. The power-on switch, a green LED indicating that power is on, the visible and audible alarms, the defeat switch, the "defeat"-position warning LED, the alarm acknowledge, and the air pressure supply connector are all conveniently located on this single panel.
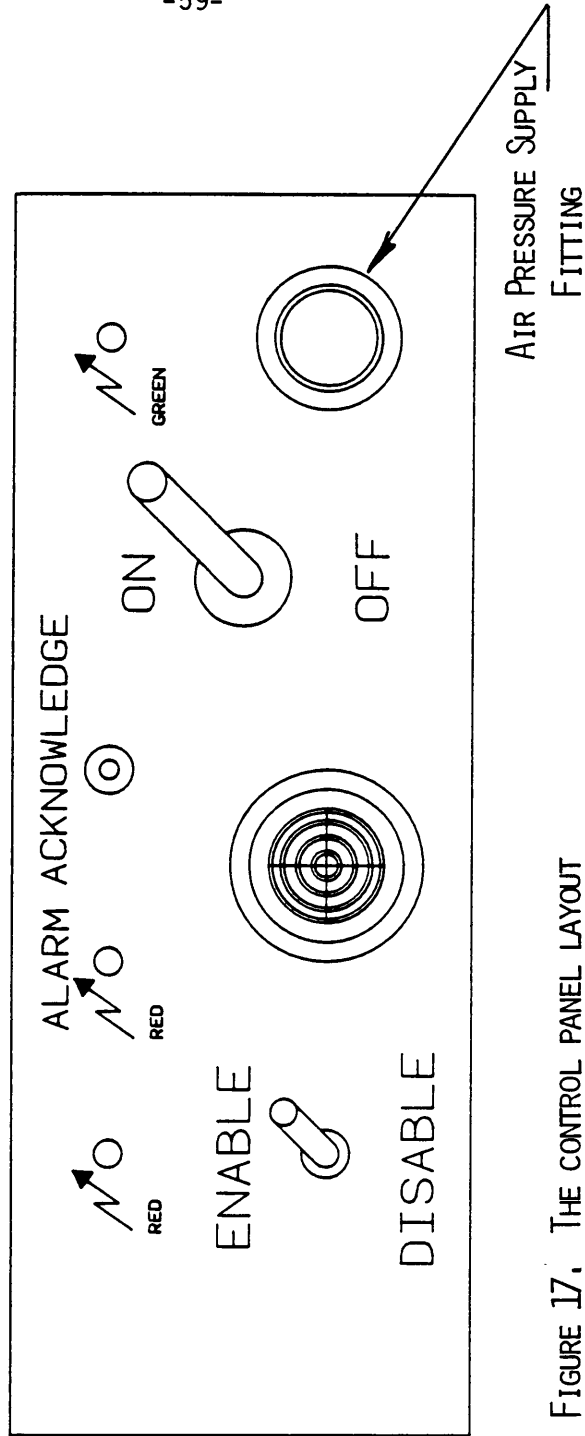
FIGURE 17. THE CONTROL PANEL LAYOUT

System Testing

The performance of a Respironics Sleep-Easy® NCPAP is evaluated and finally compared to the performance of a positive displacement-type device, rather than evaluating the positive displacement-type directly in treating apneic patients, because of the restrictions of the Food and Drug Administration guidelines for non-invasive testing and using human subjects. Since the actual clinical trials of this device are not within the scope of this development work, comparison of the performance of the proposed device with that of the current fan-type device is sufficient to show the adequacy of the new therapeutic device.

Known pressures from 0 cm of H_2O to 12 cm of H_2O as determined by an inclined manometer were used to calibrate the strain gage pressure transducer. The known pressures were supplied to the pressure transducer and the resulting deflection on a strip chart recorder noted. This calibration procedure was performed both before and after the systems' performance tests. The results of the pre-test and post-test calibration procedure were averaged for each pressure level to develop a calibration curve for the strain gage pressure transducer. The calibration curve for the strain gage pressure transducer and a schematic diagram of the testing set-up are included for completeness at the end of this thesis in Appendix A.

Each device was tested in two configurations, first with no net patient airflow and then with a resting adult's tidal respiration. Each device was

adjusted to provide 10 cm of H_2O pressure to the patient regardless of the flow demand. Results from the testing of a current fan-type NCPAP therapy system in the Bio-medical Engineering Laboratory are shown in Figs. 18 and 19. Figure 18 shows the pressure response of the system when the device is run with no net patient flow. The device was run with the nose mask blocked and the pressure monitored with a calibrated strain gage pressure transducer located just prior to the mask. As shown in Fig. 18, the pressure rises quickly to the steady-state therapeutic pressure, about 9.75 cm of H_2O in this case, in just over 2 seconds and remains very steady, fluctuating between 9.5 and 9.75 cm H_2O.

Figure 19 shows the pressure fluctuation found when the NCPAP device is required to meet the flow demands of the resting healthy adult. The patient's airway pressure ranged from a low of 9.0 cm of H_2O on inhalation, to a high of 11 cm of H_2O on exhalation. The pump's inability to supply pressurized air to fill the increased volume of the expanding lungs instantaneously brings about the slight dip in pressure found on inhalation. The slight rise in pressure on exhalation is the pressure differential necessary for exhalation through the non-rebreathing valve. Since this fan-type device is commonly and effectively used in the treatment of Sleep Apnea Syndrome, this performance is assumed adequate for evaluation of future devices.

The performance of the positive-displacement system with no net patient airflow is given in Fig. 20. With no net patient airflow, the device's output pressure rose to its steady-state pressure of 10 cm of H_2O in 4
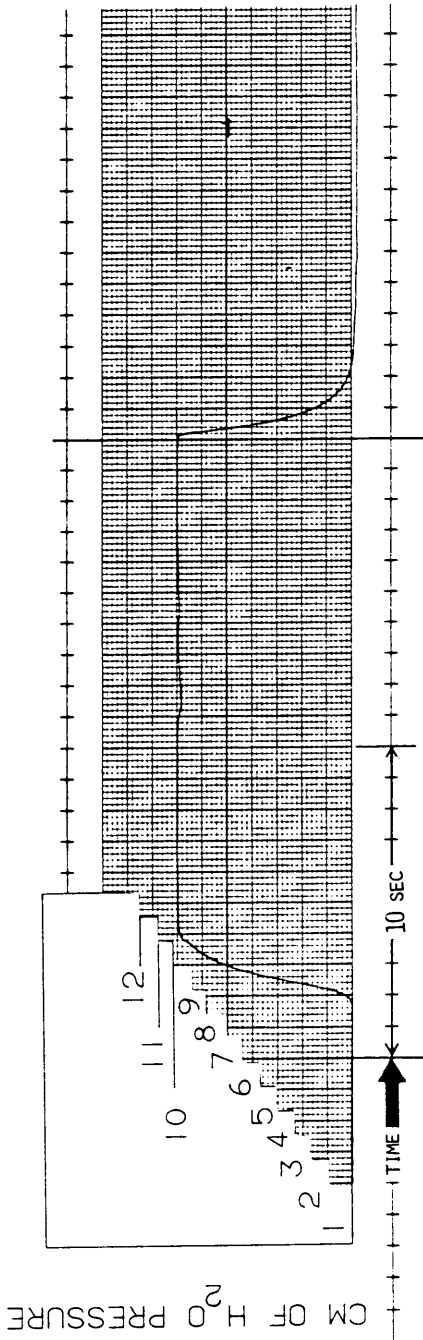
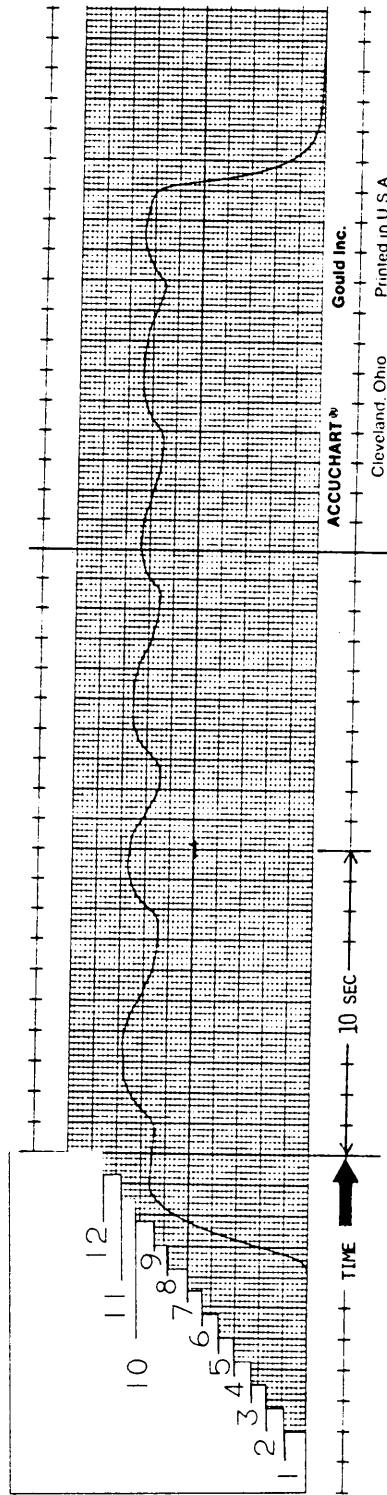FIGURE 18. RESULTS OF THE NO-FLOW PRESSURE TEST OF THE FAN-TYPE NCPAP SYSTEM

FIGURE 19. RESULTS OF THE TIDAL-FLOW PRESSURE TEST OF THE FAN-TYPE NCPAP SYSTEM
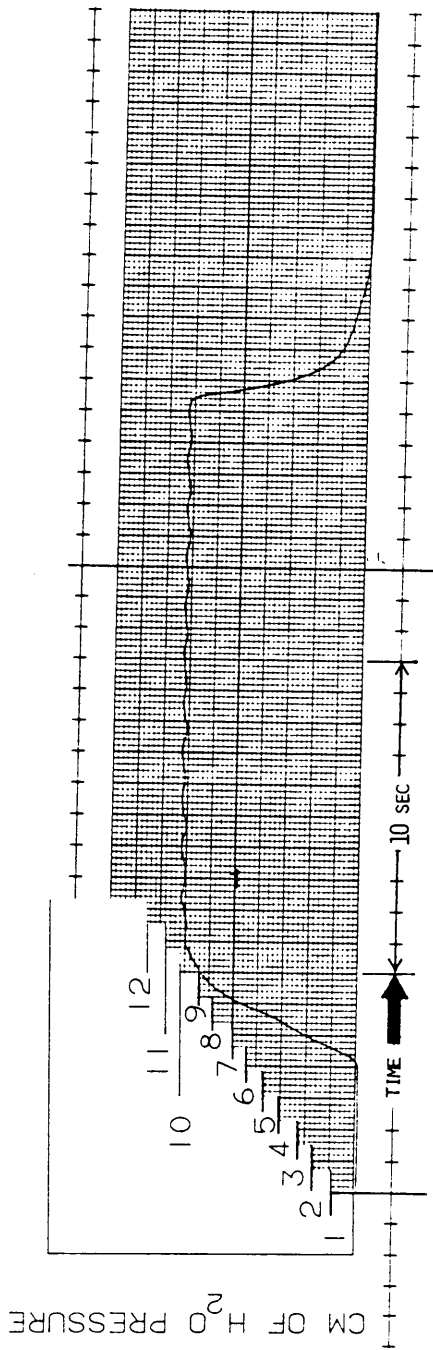
FIGURE 20. RESULTS OF THE NO-FLOW PRESSURE TEST OF THE BELLOWS-TYPE NCPAP SYSTEM

seconds. It is reasonable to expect the positive-displacement pump system to reach its steady-state condition in a longer time because of the 6.5 liter reservoir. With no patient flow the device controlled the air supply line pressure very well; the pressure ranged from a low of 9.75 cm H_2O to a high of 10.25 cm of H_22O. With a fluctuation of only 0.50 cm H_2O, the pressure was held within a 2.5% band around the steady state pressure. This is comparable to the fan-type device which held the no-flow pressure between 9.5 and 9.75 cm of H_2O.

In attempting to provide the 10 cm of H_2O pressure to meet the tidal-flow demand of the resting healthy adult, the positive-displacement pump was equally impressive. As shown in Fig. 21, the line pressure was held between 9.25 cm of H_2O and 10.75 cm of H_2O. The 9.25 cm of H_2O level was produced in the nasal mask during the inhalation phase of respiration and the 10.75 cm of H_2O pressure produced during exhalation. Again, this compares favorably with the current system which held the pressure within the range of 9.0 and 11.0 cm of H_2O.

The performance of the positive-displacement NCPAP therapy device is comparable to that of the current fan-type NCPAP system. It is believed that the proposed system will be equally successful in eliminating apneic events in adult patients with Sleep Apnea Syndrome. In addition, because of the more comfortable and effective custom nasal mask and exhalation valve, this interface should be used with all apnea treatment devices.
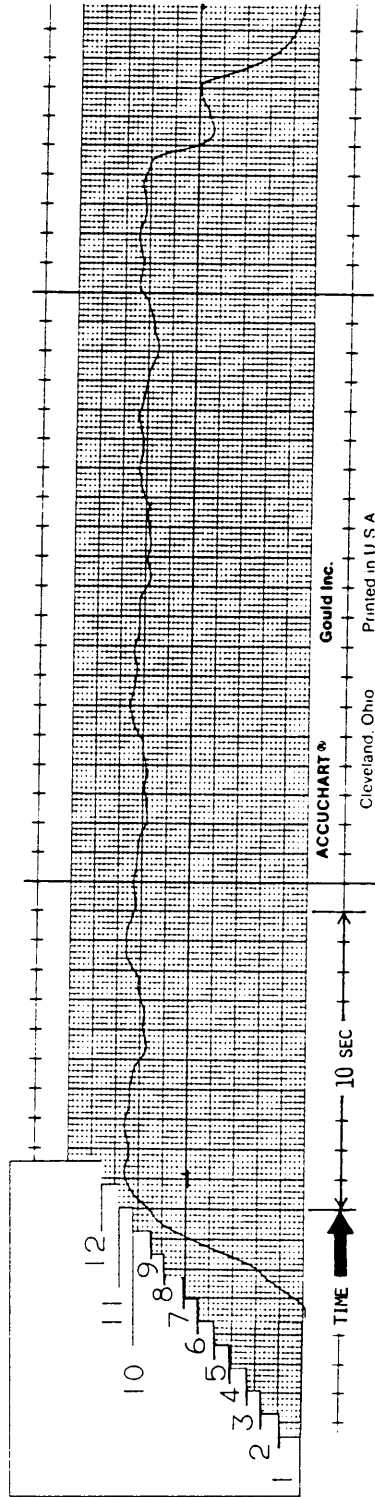
FIGURE 21. RESULTS OF THE TIDAL-FLOW PRESSURE TEST OF THE BELLOWS-TYPE NCPAP SYSTEM

Recommendations

The positive-displacement pump detailed in this thesis is a prototype for future mass production of units using the same principles. The formidable task of remodeling the unit for mass production remains. This research work has proven the feasibility of an electronically-controlled positive-displacement pump in the treatment of adult Sleep Apnea Syndrome. Since this work began, manufacturers of fan-type therapy units have begun addressing the noise and humidity problems of their devices and are taking corrective actions. As a result, Respironics recently introduced its Sleep-Easy III® model, a fan-type unit within a 1 cubic foot volume with redesigned fan and ports for quieter operation. Because of their fan-type pressure generation scheme, these units will enjoy a decided size advantage over any displacement-type therapy unit. The introduction of these small, quiet devices all but ensures that a larger displacement-type device will not gain consumer support.

This research work on a positive displacement-type pump has not been a wasted effort, however. The device detailed in this thesis contains features that the author believes should be recognized and included in future therapy units -- whether they be fan or displacement type. Alarm circuitry and a more comfortable and effective nasal mask should be included in future SAS therapy devices. For this reason, it is recommended that funding be provided in order for development work to continue on these elements of this new device. Future development work should include the testing of a

variation of NCPAP therapy, and the construction and complete clinical testing of a second generation pressure controlled fan-type pump which includes the motor control circuitry, alarm circuitry, and the custom nasal mask and exhalation valve assembly of the displacement-type pump. This device has the potential to further lower the noise levels of the newest and quietest models by running the fan at a slower speed when appropriate and eliminating the noise produced by the constant pressure bleed-off leak.

Physical design changes necessary before production could be instituted include the development of a more encompassing alarm system and a less expensive sensor, and the inclusion of Dr. Arp's humidifier. The alarm system present on the prototype device provides the minimum level of patient safety. Modifications should be done on this system to provide a selectable alarm threshold and apnea detection. It may be desirable also to include a microprocessor-based monitoring system in some models. With this, long term or follow-up sleep studies may be performed at home and the data saved for later analysis. Reliable compliance studies would be easily performed with such a system.

Replacement of the brass diaphragm sensor is necessary for this to occur. The sensor found in the prototype device would be a prohibitive cost to the end product. Complete testing of a low-cost sensor, perhaps based on the operation of a silicone balloon, should be completed. One such variation was successfully tested in the Bio-medical Engineering Laboratory. Inclusion of an air humidification device to the main NCPAP therapy device

is essential. Dr. Arp's humidifier includes the flexibility of varying droplet size for either vaporization or nebularization of the water, for selection of the relative humidity of the air, and for the cyclic administration of this humidification. This device should provide humidification to meet any patient's needs.
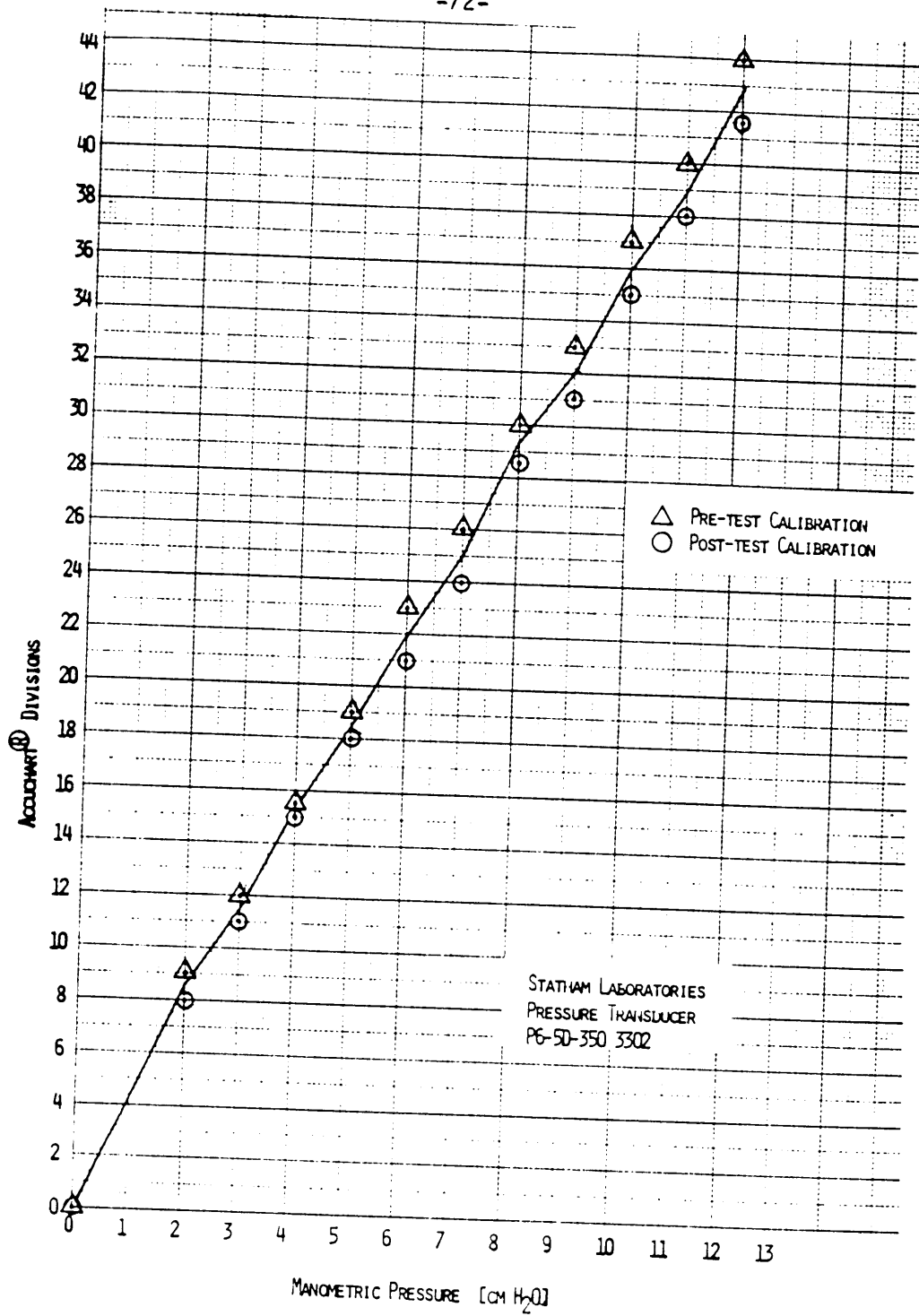
Finally, the continued development of a comfortable and effective patient interface with a minimum dead space to reduce re-inhalation of exhaled gases should be supported. The nasal mask and exhalation valve detailed here is one such interface. An intranasal balloon that could seal the airway and surround a standard nasal cannula pressure supply line may also be feasible and should be explored. In the meantime, the production of custom nose masks should be investigated. A quick, easy, and reliable method for constructing the masks should be developed. Presently, the most promising method appears to be the manufacture of 3 to 4 standard-sized nasal shells for custom lining in a physician's office with a resilient form fitting material like Co-Soft® or Tru-Soft® dental reliner. Once instructed how to reline the mask, the patient could handle the job at home. This relining material is easy to work with and can be stripped out and replaced when cleaning the mask or when it becomes uncomfortable.

A comprehensive study on patient compliance and its effect on Nasal Continuous Positive Airway Pressure therapy should also be performed. Subjective evaluations of patient/machine interfaces, machine type and configuration, safety features, and air humidity are also recommended. Since
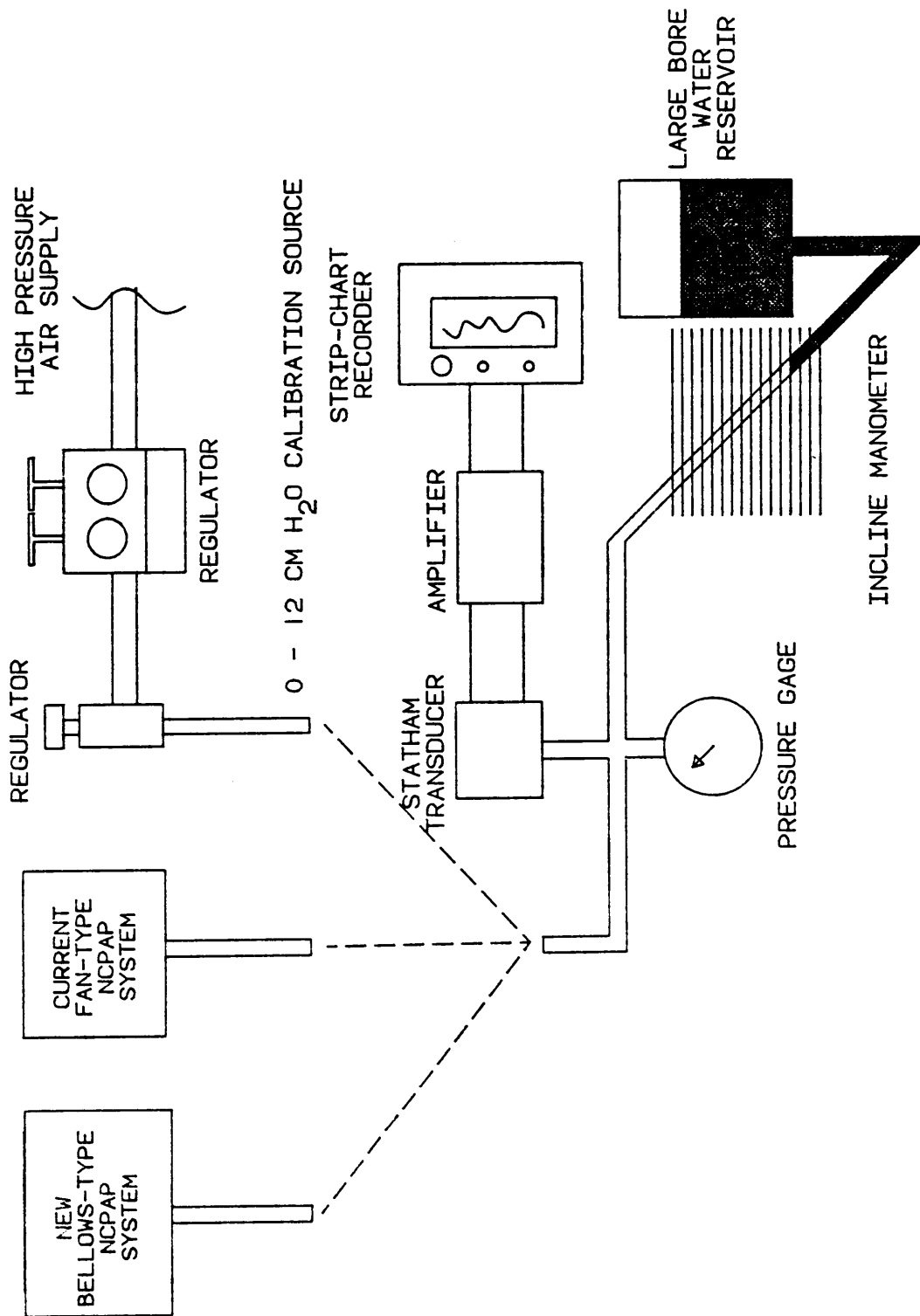
the true mechanism for NCPAP therapy is not known, an investigation into the effectiveness a variation of NCPAP therapy with lower airway pressure during exhalation and pause is recommended. It may be possible that airway pressure need only be applied at the start of inhalation, where the opportunity for tracheal blockage is greatest. If effective, this variation would be a significant improvement in NCPAP therapy because the lower pressures would increase cardiac output and venous return. In addition, this could lead to even quieter therapeutic devices.

APPENDIX A

A1. PRESSURE TRANSDUCER CALIBRATION CURVE

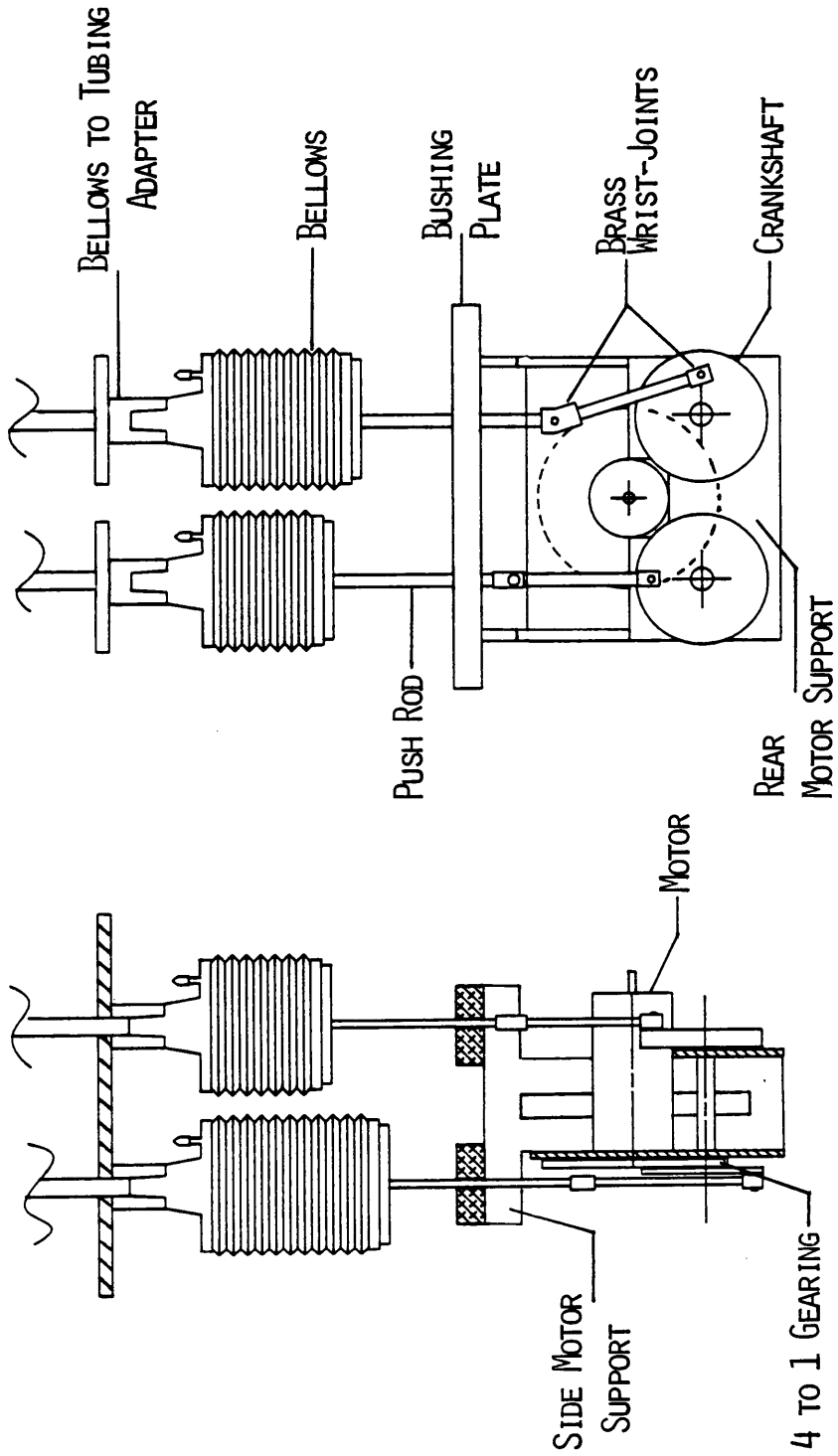A2. SCHEMATIC DIAGRAM OF THE TESTING SET-UP

Appendix B

BELLOWS TO TUBING ADAPTER

BELLOWS

BUSHING PLATE

BRASS WRIST-JOINTS

CRANKSHAFT

PUSH ROD

REAR MOTOR SUPPORT

MOTOR

SIDE MOTOR SUPPORT

4 TO 1 GEARING

B1. MECHANICAL DEVICE ASSEMBLY

NOTE
DRAWING NOT TO SCALE
ALL DIMENSIONS IN INCHES

DRILL NO. 11 THRU

6.50

6.00

5.50

3.45

2.00

0.50

0.35

0.375

1.25

0.188

2.92

2.62

4.25

5.58

5.87

7.25

8.31

8.50

DRILL 1 1/4 THRU

DRILL NO. 21 THRU
TAP 10-32
4 PLACES

DRILL NO. 11 THRU
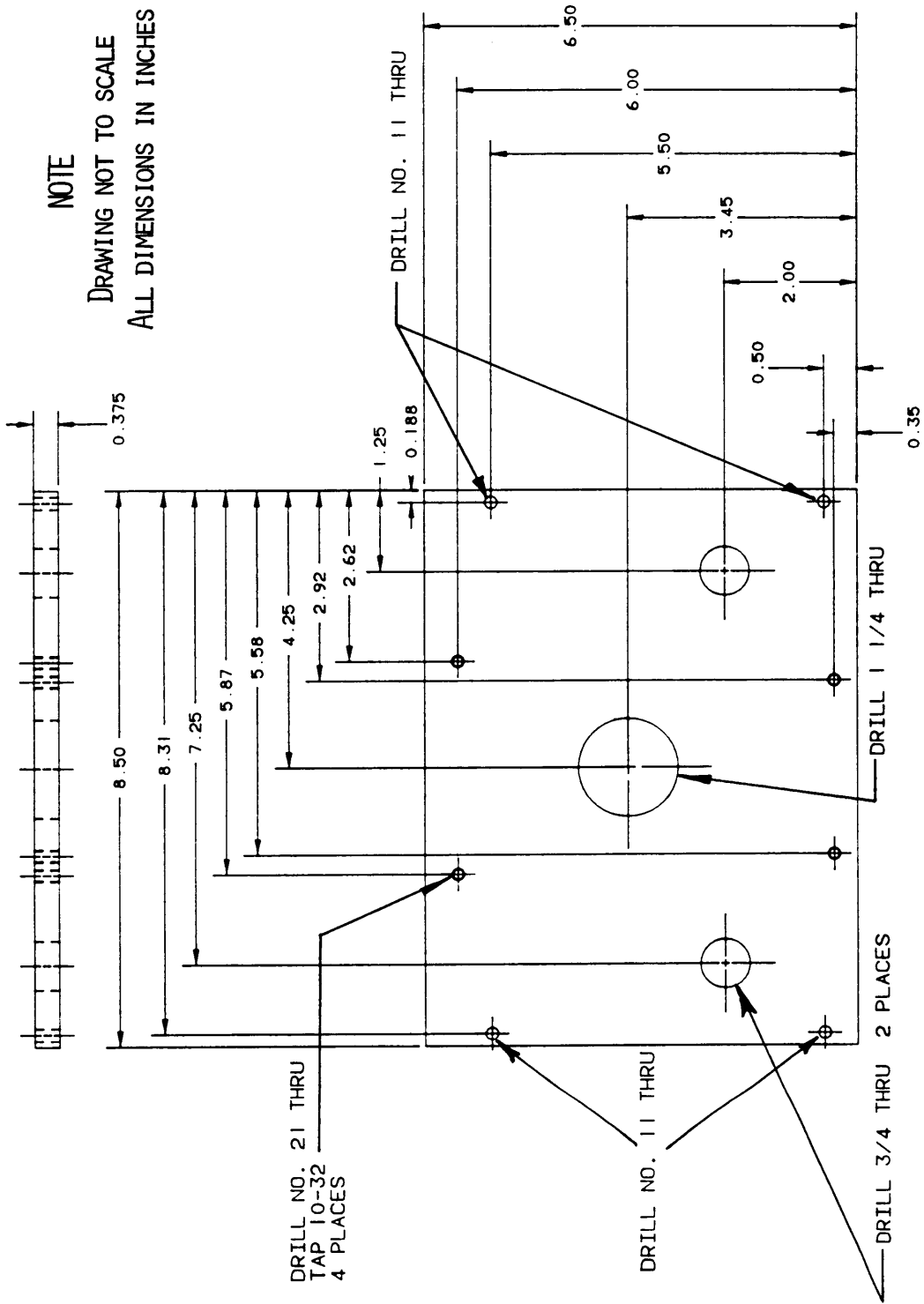
DRILL 3/4 THRU 2 PLACES

B2, FRONTAL MOTOR SUPPORT

NOTE
DRAWING NOT TO SCALE
ALL DIMENSIONS IN INCHES

DRILL NO 11 THRU
CSK 82° TO 0.36 DIA
4 PLACES

1/4 MILL X 1/2 DEEP
4 PLACES

DRILL 1.25 THRU

6.00

5.625

5.36

3.25

2.70

0.375

0.375

0.672

2.00

3.33

3.62

4.00

0.188

B3. MOTOR MOUNT PLATE

NOTE
DRAWING NOT TO SCALE
ALL DIMENSIONS IN INCHES

7.109

0.75

DRILL NO. 21 X 1 DEEP
TAP 10-32 X 3/4 DEEP
6 PLACES

7.869

5.672

4.672

3.188

2.188

1.00

7.00

8.00

2.50

6.00

7.50

0.188

0.375

B4. SIDE MOTOR SUPPORT

NOTE

DRAWING NOT TO SCALE

ALL DIMENSIONS IN INCHES

DRILL NO. 11 THRU
4 PLACES

3.00

2.00

1.25

0.75

0.50

0.375

3.25

2.50

5.25

6.00

7.25

8.31

8.50

1.25

0.188

1/4 MILL X 3/4
2 PLACES

DRILL 3/4 THRU
2 PLACES

B5, REAR MOTOR SUPPORT

NOTE
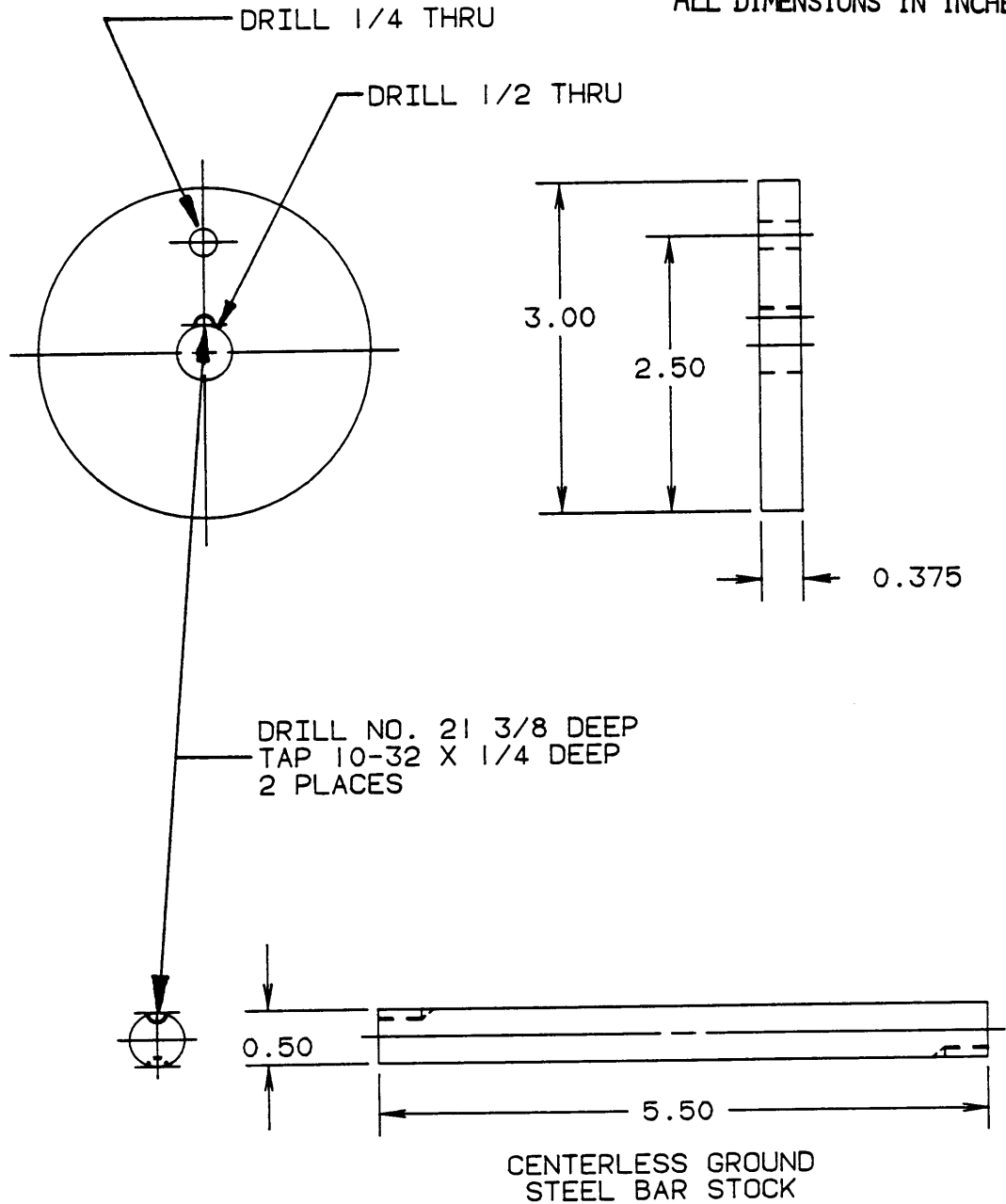DRAWING NOT TO SCALE
ALL DIMENSIONS IN INCHES

3.50

0.375

2.75

0.75

1.55

2.35

DRILL NO. 11 THRU
2 PLACES

B6. REAR SUPPORT ADJUSTOR

NOTE
DRAWING NOT TO SCALE
ALL DIMENSIONS IN INCHES

DRILL 1/4 THRU

DRILL 1/2 THRU

3.00

2.50

0.375

DRILL NO. 21 3/8 DEEP
TAP 10-32 X 1/4 DEEP
2 PLACES

0.50

5.50

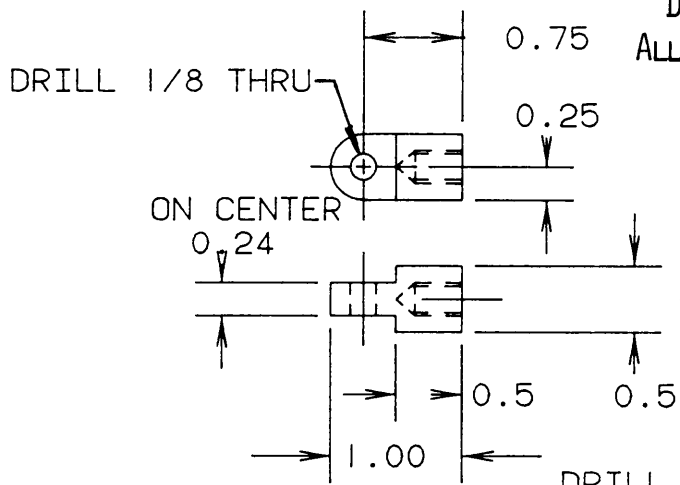CENTERLESS GROUND
STEEL BAR STOCK

NUMBER REQUIRED: 2 EACH DETAIL

B7. CRANKSHAFT ASSEMBLY

NOTE
DRAWING NOT TO SCALE
ALL DIMENSIONS IN INCHES

DRILL 1/8 THRU

0.75

0.25

ON CENTER

0.24

0.5   0.5

1.00

DRILL NO. 7 1/2 DEEP
TAP 1/4-20 3/8
ON CENTER
1 PLACE EACH DETAIL

DRILL 1/8 THRU

0.75

0.25

ON CENTER

0.25

0.5

0.5

1.00

DRILL 9/32 THRU

0.25

0.5

0.75

1.00
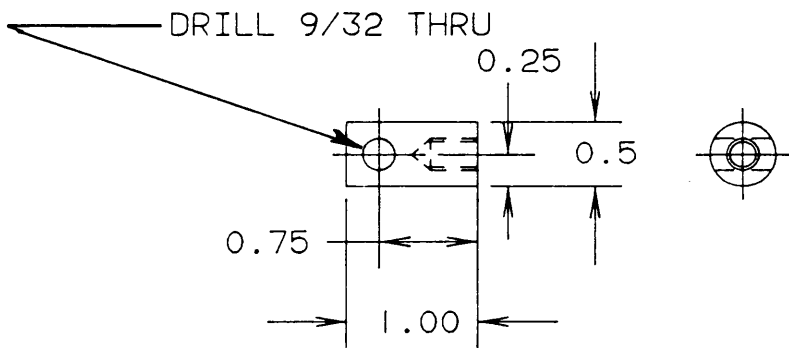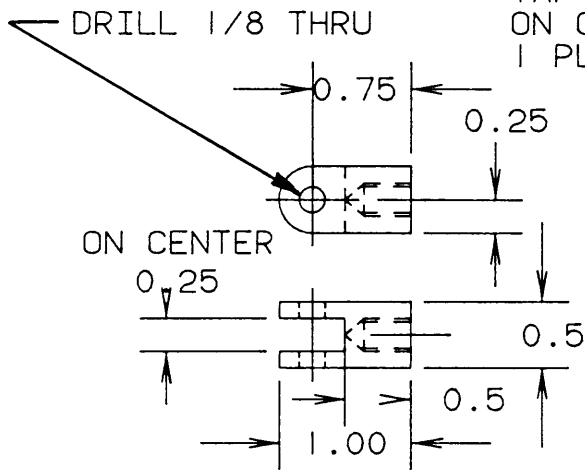
QUANTITY REQUIRED: 4 OF EACH DETAIL

B8. BRASS WRIST-JOINTS

DRILL 1/2 THRU 2 PLACES

DRILL NO. 11 THRU 2 PLACES

1.70

0.75

0.85

3.00

1.94

9.00

10.06

12.00
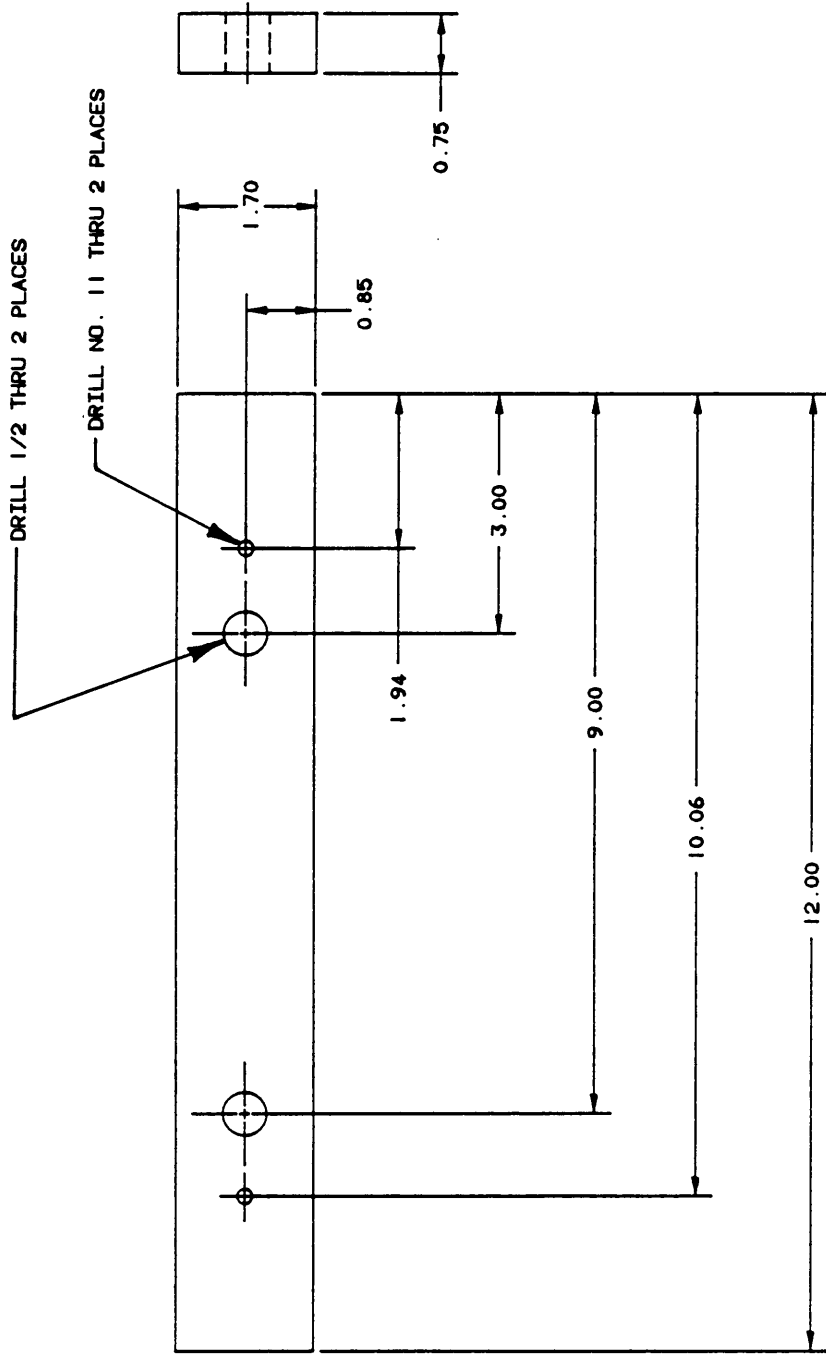
NOTE
DRAWING NOT TO SCALE
ALL DIMENSIONS IN INCHES

B9, BUSHING PLATE
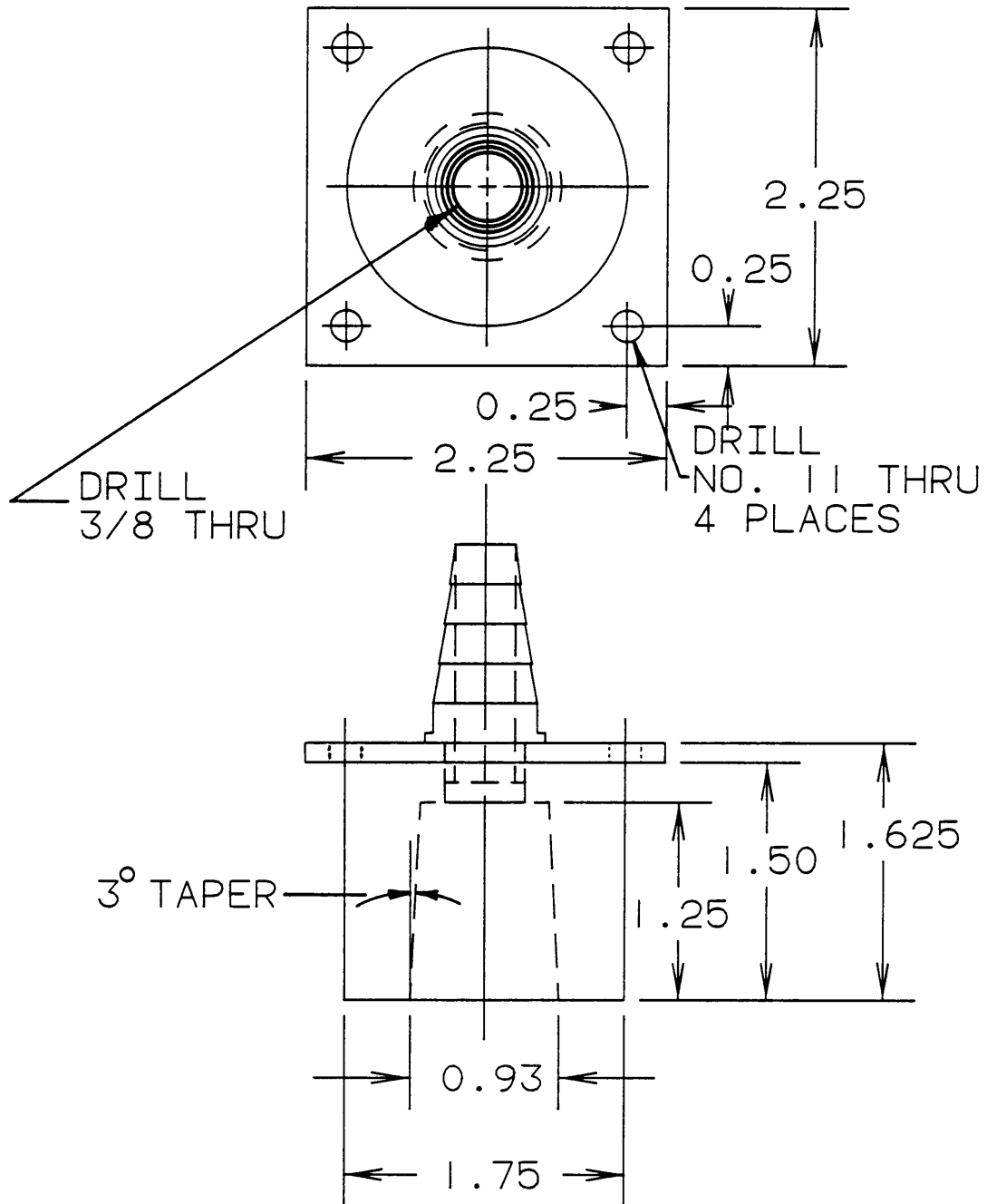
2.25

0.25

DRILL
3/8 THRU

0.25

2.25

DRILL
NO. 11 THRU
4 PLACES

3° TAPER

1.50   1.625

1.25

0.93

1.75

B10, BELLOWS TO TUBING ADAPTER

3.75

DRILL NO. 11 THRU
4 PLACES

NOTE
DRAWING NOT TO SCALE
ALL DIMENSIONS IN INCHES

4.25

9.175

3 POUND
COUNTER WEIGHT

3.37

3.50
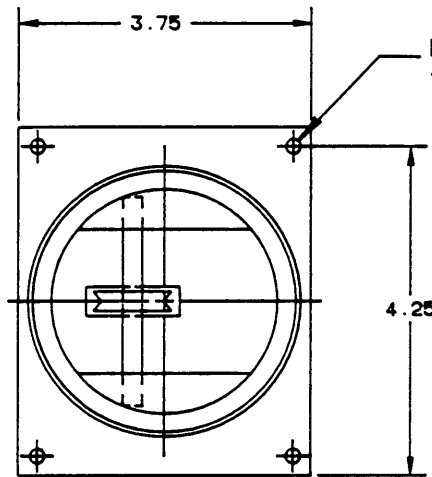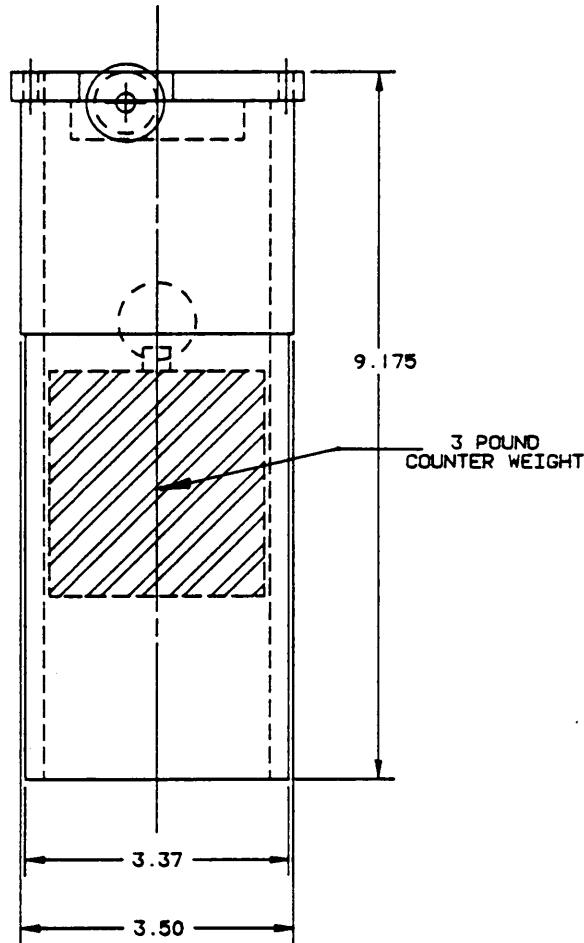
B11, COUNTERWEIGHT CYLINDER

BAKELITE® RESERVOIR PANELS
0.125 IN. THICKNESS

DRILL NO. 11 THRU
12 PLACES
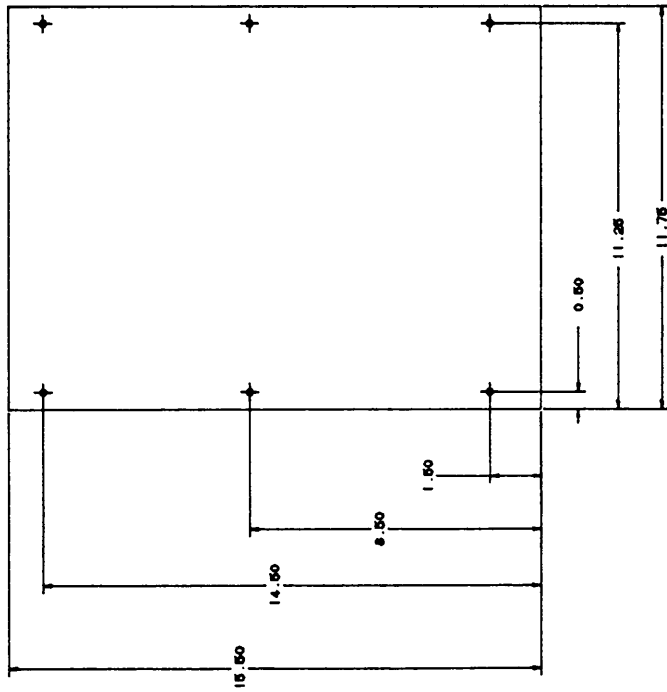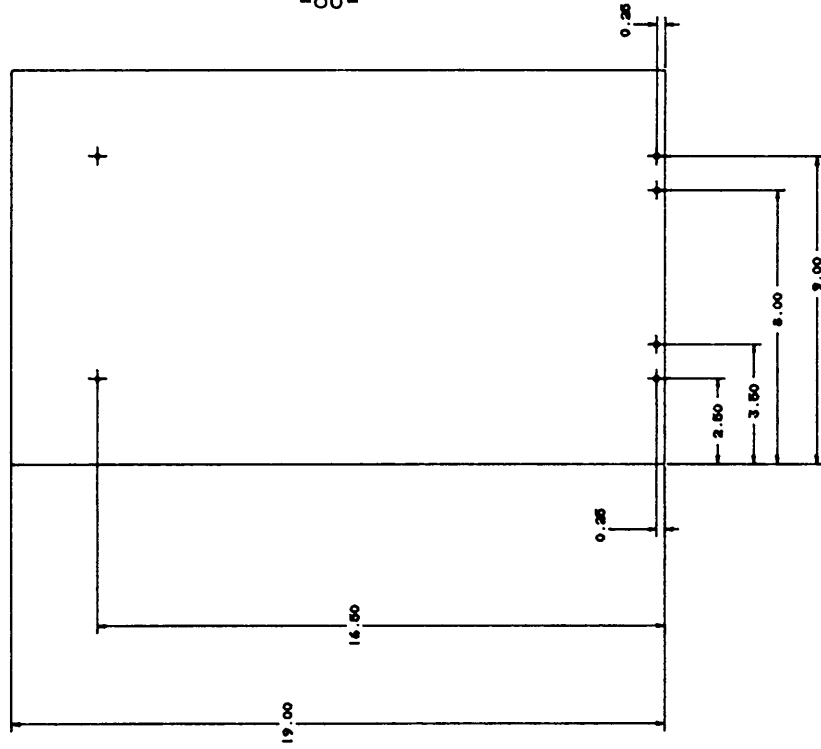
B12, BAKELITE RESERVOIR SUPPORT

B13. English to metric dimension conversion chart

English dimension [in]	SI equivalent [mm]
0.125	3.2
0.1875	4.8
0.24	6.1
0.25	6.4
0.2813	7.1
0.35	8.9
0.36	9.1
0.375	9.53
0.4	10.16
0.5	12.7
0.5625	14.29
0.672	15.93
0.75	19.1
0.85	21.6
0.93	23.6
1.25	31.8
1.5	38.1
1.55	39.4
1.625	41.3
1.7	41.5
1.94	49.3
2.00	50.8
2.1875	55.6
2.25	57.2
2.35	59.7
2.50	63.5
2.62	66.6
2.675	67.95
2.70	68.6
2.75	69.9
2.92	74.2
3.1875	80.96
3.25	82.6
3.33	84.6
3.37	85.6
3.45	87.6
3.5	88.9
3.50	88.0
3.75	95.3
4.00	101.6
4.20	106.7
4.25	108.0
4.672	118.7

English dimension [in]	SI equivalent [mm]
5.00	127
5.25	133.4
5.36	136.1
5.5	139.7
5.58	141.7
5.62	142.7
5.672	144.1
5.87	149.1
6.000	152.4
6.50	165.1
6.62	168.1
7.00	177.8
7.109	178.3
7.25	184.2
7.869	199.9
8.0	203.
8.31	211.1
8.5	215.9
9.0	229
9.175	233
10.06	255.5
11.25	285.8
11.75	298.5
12.00	304.8
12.7	322.6
14.00	355.6
14.5	368
15.5	394
16.00	406.4
16.5	419
19.0	483

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