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A Review of Dissolved Gas Supersaturation Literature

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A Review of Dissolved Gas Supersaturation Literature

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Abstract

Dissolved gas supersaturation is a condition that results from natural and human-caused processes. Supersaturation can result in gas bubble disease which has been described in a wide variety of fishes and invertebrates. In recent years dissolved gas supersaturation resulting from dams and thermal discharges has produced mortalities of fish in several cases. This review discusses most of the available literature dealing with dissolved gas supersaturation and the recorded cases of gas bubble disease.

Gas bubble disease is a condition that affects aquatic animals residing in fresh or marine waters that are supersaturated with atmospheric gases. Supersaturation, and the gas bubble disease that may result in aquatic organisms, are not recent discoveries nor are they only caused by human activities. However, only in recent years has supersaturation become a problem of sufficient magnitude to draw widespread attention and concern.

The majority of research dealing with dissolved gas supersaturation has been stimulated by a problem of considerable magnitude that was observed in the Columbia River system beginning in the 1960's. More recently, interest has been further stimulated by the discovery of deleterious effects of supersaturation resulting from thermal effluents.

This review is an attempt to provide a greater dissemination of the available existing knowledge regarding dissolved gas supersaturation and the resulting gas bubble disease. The review discusses the causes of supersaturation, the organisms affected by supersaturation, factors affecting susceptibility of aquatic organisms to gas bubble disease, and various other related topics. The knowledge of this subject is considerable as evidenced by the length of this review. Many important questions remain to be answered. This is particularly true regarding the application of laboratory results to conditions faced by aquatic organisms under natural

conditions. Much remains to be learned about the physiological aspects of gas bubble disease.

In order to understand gas bubble disease and its cause, it is necessary to be familiar with the physical laws governing dissolved gases and the factors that determine the level of supersaturation. Boyer (1974), Woelke et al. (1974), and Harvey (1975) discussed the solubilities of dissolved gases in water as they relate to gas bubble disease.

Harvey (1975) provided an excellent discussion of this subject for those not familiar with the physical laws describing the solubilities of gases in a liquid. The solubility of atmospheric gases in water is determined by the water's dissolved solids content, characteristics of the various gases, the total pressure, and the water temperature. Although total dissolved solids can affect solubility, this is not a significant variable in most fresh waters but must be considered as a significant variable in marine waters.

The atmospheric gases of importance are nitrogen, oxygen, and argon. These gases are present in air at partial pressures of approximately 78% nitrogen, 21% oxygen, and 1% argon. Nitrogen and argon are normally considered together because both are biologically inert gases while oxygen is a biologically active gas.

The solubility of each gas is determined by the mass of the individual gas and its partial pressure in the atmosphere. Oxygen (21%) has

only one-fourth the partial pressure of nitrogen (79%) in the atmosphere, but is twice as soluble as nitrogen. Therefore, in water, oxygen (35%) is one-half as plentiful as nitrogen (65%) (Harvey 1975).

The major environmental factors that affect solubility are pressure and temperature. According to Henry's Law, the mass of a gas dissolved in a liquid at a constant temperature is proportional to the pressure exerted on the solvent. Thus, as the pressure on a given volume of water increases, the capacity of that volume of water to hold dissolved gas also increases. Pressure is increased in water by hydrostatic head. Hydrostatic pressure increases rapidly with depth, greatly increasing the capacity of deeper water to hold dissolved gas as compared to shallow water.

The capacity of water to hold dissolved gas is inversely related to temperature. As the temperature of a volume of water increases, the volume of dissolved gas it will hold at equilibrium decreases. Thus, increasing water temperatures will produce supersaturation in water that is initially saturated.

This brief review of the factors affecting solubility only discusses a few of the major variables. Those interested in a more detailed review of the subject should refer to Harvey (1975) or to texts describing the gas laws in detail.

History of Gas Bubble Disease

Early Observations

There are several nineteenth century records of what appears to have been gas bubble disease. Hoppe-Seyler (1857), Bert (1873), and Regnard (1884) recorded external signs in fish that apparently represent gas bubble disease. The first complete description of gas bubble disease and its cause was provided in a series of papers resulting from an air supersaturation problem at the United States Bureau of Fisheries station at Woods Hole, Massachusetts (Gorham 1898, 1901; Marsh 1903, 1910; Marsh and Gorham 1905). This series of initial papers dealt with mortality, signs, and experiments to determine and correct the cause of the disease in aquarium fish.

The first description of the outward signs of gas bubble disease was given by Gorham (1901). Vesicles (gas bubbles or blisters) were found in the fins and other external surfaces of several

marine fishes. Bubbles frequently occurred behind the cornea and in the loose connective tissues of the eye, producing a severe exophthalmia or "pop-eye" condition. Bubbles were found less frequently in the gills, lining of the mouth, or along the lateral line of exposed fish. These bubbles gradually increased in size as the length of exposure to supersaturation increased. Fish with these signs also showed loss of equilibrium. The time to death varied from several hours to several weeks following the appearance of detectable bubbles.

Marsh and Gorham (1905) further described internal signs and lesions of the disease. Free gas bubbles (or emboli) within blood vessels were observed. The amount varied from a few scattered bubbles to complete occlusion and distention of the vessels. The walls of the auricle and ventricle were often emphysematous. In some fish, the auricle was filled with gas even though it continued to beat. The main vessels of the gills contained gas bubbles. Gas in the gill filaments was described as the most constant and significant lesion of gas bubble disease. Death of the fish was attributed to stasis of the blood caused by emboli.

In addition to observations of fish, Gorham (1901) also reported signs of the disease in squid, bivalve mollusks, scallops, hydroids, squid egg-sacs, and green algae. No detailed discussion of the disease in these organisms was given by Gorham.

Gorham (1901) experimentally produced gas bubble disease in fish held in closed containers by reducing the pressure. He also was able to cause the signs to disappear by subjecting fish to a pressure comparable to that exerted by a water depth of 4.9 m. He concluded that gas bubble disease was caused by a reduction in the pressure to which fish normally were subjected. Marsh and Gorham (1905) later corrected this mistaken conclusion.

Gorham (1901) reported that whereas fish in shallow aquaria developed gas bubble disease, fish held in ponds 2–4 m deep with water from the same source remained free of it. This was the first indication of the major difference between artificially shallow water conditions and the more natural situations that permit hydrostatic compensation.

Later, Marsh and Gorham (1905) discussed the solubility of gases in water and the relationship of respiratory processes to gas bubble dis-

ease. They concluded that the disease was caused chiefly, if not solely, by excessive dissolved nitrogen gas. This conclusion was based on the analysis of bubbles from tissues and blood vessels of animals with gas bubble disease. These bubbles contained 92% to 97% nitrogen, the remainder being oxygen. Bubbles formed in the supersaturated water had nitrogen and oxygen in the same ratio as found in air. Marsh and Gorham concluded hemoglobin has the capacity to modify the effect of oxygen by removing it from the dissolved state.

Marsh and Gorham (1905) reported several instances of naturally occurring supersaturated fresh waters. Rainbow trout (*Salmo gairdneri*) in these waters showed the same signs of the disease as the marine fish from Woods Hole. Experimentally they determined that trout and some cyprinids have nearly equal susceptibility to gas bubble disease, whereas goldfish (*Carassius auratus*) are not affected by the same levels of supersaturation.

The prevention of the disease by removal of excess dissolved gases through aeration was discussed by Marsh and Gorham (1905). The supersaturation condition at Woods Hole was corrected by replacing an intake pipe that had allowed air to be sucked into the water supply. Marsh (1910) later removed excess gases by trickling water over stacks of perforated shallow pans.

In this series of papers, Marsh and Gorham established the basic knowledge of gas bubble disease. Most subsequent investigations have confirmed and expanded on their work. Anyone seriously interested in the problem would be well advised to read these early works, in particular, Marsh and Gorham (1905).

During the 40 years following the work by Marsh and Gorham a few scattered reports of gas bubble disease appeared in the literature. Shelford and Allee (1913) encountered it in experiments to test the reaction of fish to gradients of atmospheric gases. Supersaturation was produced by raising the temperature of water about 9 C. This study was designed to achieve other objectives and made no new contributions to the understanding of the disease.

Plehn (1924) reported the occurrence of gas bubble disease due to supersaturation brought about by photosynthetic activity. Dissolved nitrogen concentrations were not measured but those of dissolved oxygen were three times sat-

uration. This situation occurred during winter conditions where photosynthetic activity occurred under clear ice, producing excess gas that could not escape to the atmosphere. Wiebe and McGavock (1932) experimentally exposed a variety of fishes to dissolved oxygen concentrations two to three times saturation for periods of 20 to 50 days. No signs of the disease were found in these fish even though evidence of it was sought.

Mrsic (1933) observed that fish reared in tap water suffered gas bubble disease when oxygen and nitrogen concentrations were below saturation but carbon dioxide was supersaturated. Mrsic reported 75% of fish in water with 138 mg/liter carbon dioxide suffered gas bubble disease whereas no fish in water having 135 mg/liter carbon dioxide showed signs of it. Although Mrsic attributed the disease to carbon dioxide it appears unlikely that a rise of only 3 mg/liter at those high concentrations would produce such a high incidence of the disease.

Embrey (1934) reported gas bubble disease in trout fry at the Cornell University hatchery. The fry were hatched in water that had been heated to raise the temperature 5 C. Unhatched eggs were apparently unaffected whereas the yolk sacs of newly hatched fry were distended by gas bubbles. The disease was attributed to excess dissolved nitrogen and was prevented through aeration, which was accomplished by passing the water over a series of baffles.

Woodbury (1941) described a sudden mortality of fish showing signs characteristic of gas bubble disease in a Wisconsin lake. Bubbles were observed in the gills of these dying fish as well as between fin rays and under scales. The disease was attributed to dissolved oxygen levels in excess of 300% saturation. This mortality followed an extensive algal bloom during a period of sunny weather.

Rucker and Tuttle (1948) noted a hatchery water supply at Leavenworth, Washington, was supersaturated with nitrogen. Fish subjected to this water were reported to suffer gas bubble disease although few details are given.

In the late 1940's, the disease was encountered in larval marine fish at a Swedish hatchery (Dannevig and Dannevig 1950). Larval herring (*Clupea harengus*) and flatfish developed bubbles in the intestine. The flatfish larvae were able to pass the bubbles through the anus while the herring were unable to do so and suffered

extensive mortalities. The cause of the supersaturation was air leaks in the water supply system. Henly (1952) described differences between physoclistous and physostomous fish larvae with the disease at this hatchery. She also noted that oyster larvae suffer great mortality when subjected to supersaturated water. Supersaturation in the Swedish hatchery water supply was corrected by passing the water through two sand filters.

Several cases of gas bubble disease were reported in the 1950's. Rucker and Hodgeboom (1953) described the disease in salmonid yolk sac fry reared in a spring water supply which had oxygen at 70% of saturation and nitrogen at 120% of saturation. Supersaturation was reduced by passing the water through an 18-m-long agitation weir.

Matsue et al. (1953) reported the disease in fishes held in supersaturated water supplies in Japan. Fish mortalities occurred in waters having nitrogen levels from 117% to 158% of saturation. Fish reportedly could not live in water with nitrogen levels above 130% of saturation. Satomi (1955) indicated there was no fear of gas bubble disease in adult trout reared in spring water that had dissolved nitrogen levels under 120% of saturation.

Renfro (1963) attributed the death of numerous marine fishes in Galveston Bay, Texas, to this disease. The mortality occurred following a period of calm sunny weather and intense photosynthesis. On the day following the mortality, dissolved oxygen concentrations were 250% of saturation.

These early instances of gas bubble disease were very minor in size and duration. In the Columbia River system, the disease has been recognized as a serious long-term problem affecting a large area and numerous fish.

Columbia River System

In the mid-1960's, it gradually became evident that a serious dissolved gas problem existed in the Columbia River system. Westgard (1964) observed adult chinook salmon (*Oncorhynchus tshawytscha*) suffering gas bubble disease at the McNary Spawning Channel in 1962. In this case, supersaturation was caused by the spawning channel intake. A dissolved nitrogen level of 119% of saturation was measured in an area of the channel where the fish spent considerable time.

Pauley et al. (1966) and Pauley and Nakatani

(1967) described the occurrence of the disease during 1965 in juvenile chinook salmon held in aquaria at Rocky Reach Dam on the mid-Columbia River. These papers do not describe the source of the aquarium water which probably was supersaturated river water.

The first indication that a serious supersaturation problem existed throughout the Columbia River system was provided by Ebel (1969) and Meekin (1971). Monitoring of dissolved gas levels in the Columbia and Snake rivers during high-flow periods showed that levels of 120% to 130% saturation occurred in 1966 and 1967. Migrating juvenile chinook salmon which were examined at Priest Rapids Dam on the mid-Columbia River during 1966 showed signs of gas bubble disease. Considerable increases in the migration time of these fish resulted in long periods of exposure to supersaturation (Raymond 1968, 1969). The increased length of exposure coupled with the high levels of supersaturation apparently were responsible for the appearance of the disease in the migrating juveniles. Live-cage studies with juveniles at Priest Rapids Dam in 1967 also indicated the same problem. Adult salmonids were also observed for signs at several lower Columbia River dams in 1967; a small number of them showed indications of the disease.

In 1968, signs of gas bubble disease were again observed in juvenile salmonids in the lower Columbia River (Beiningen and Ebel 1970). High mortalities occurred among juveniles held for inspection at The Dalles Dam, where, at times, approximately half the fish showed signs of the disease. In addition, there were several mortalities of adult salmonids showing similar signs downstream from the recently completed John Day Dam. At John Day Dam in 1968, all water passing the dam traveled over the spillway before the turbines were installed. This situation produced dissolved nitrogen saturations of 123–143% downstream from the dam. Problems with fish-passage facilities further complicated the situation, causing delays of migrating adult salmonids in the highly supersaturated water. It was estimated that over 20,000 summer chinook salmon were lost in this area during this episode.

Ebel (1971) and Raymond (1970) reported mortalities and signs of gas bubble disease in both juvenile and adult salmonids in the Snake River during 1970. According to Ebel, dissolved nitrogen levels in the river water ranged

from 120% to 140% of saturation for well over a month. Juvenile chinook salmon held in live-cages at Ice Harbor Dam suffered severe mortalities during this period.

Meekin and Allen (1974c) estimated that 6% to 60% of adult salmonids in the middle region of the Columbia River died between 1965 and 1970. Carcasses of adult salmon were found in the river when N₂ supersaturation reached 120% or higher. Few carcasses were found when nitrogen saturations did not exceed 112%.

A general review of the supersaturation problem in the Columbia River system through 1970 was presented by the Environmental Protection Agency (USEPA 1971). That brief review was prepared prior to completion of the many reports available today.

Dissolved gas levels in the Columbia River system between 1965 and 1969 were given by Beiningen and Ebel (1971). In each of these years, dissolved gas levels in excess of 120% of saturation were measured. At times, dissolved nitrogen saturations exceeded 140%.

May (1973) described mortalities due to gas bubble disease in 1973 below the recently constructed Libby Dam in the upper reaches of the Columbia River system. All mountain whitefish (*Prosopium williamsoni*) and cutthroat trout (*Salmo clarki*) held in shallow live-cages were dead within 4 days at total dissolved gas saturations above 130%. When average total gas levels dropped below 120%, mortality rates dropped markedly. Most fish, in an area of the river exceeding 130% of saturation, showed signs of the disease, whereas fish collected from a downstream area with saturations of 105–118% showed no sign of it.

Raymond (1979) reviewed the history of salmon migrations in the Columbia and Snake rivers between 1964 and 1975. This review includes descriptions of dissolved gas supersaturation problems during these years and improving dissolved gas conditions by 1975. The supersaturation problem in the Columbia River system has since been essentially eliminated. Ebel (1979) stated that in the Columbia and Snake rivers "... fishery agencies believe the problem of supersaturation and corresponding losses of fish to gas bubble disease is solved."

Other Recent Observations

Wyatt and Beiningen (1971) encountered gas bubble disease in trout and salmon juveniles at

a hatchery on the South Santiam River in Oregon. As in the Cultus Lake Hatchery episode (Harvey and Smith 1961), supersaturation was due to conditions that permitted air to be sucked into the water supply. Apparently most of these fish died within hours when subjected to supersaturation approaching 150%.

Although supersaturation at hydroelectric projects normally results from spilling water, MacDonald and Hyatt (1973) reported supersaturation was caused by air vented into turbines. Atlantic salmon (*Salmo salar*) and American eels (*Anguilla rostrata*) suffered gas bubble disease below the Mactaquac Dam on the St. John River, New Brunswick. An estimated 200 Atlantic salmon were killed in this incident. Nitrogen saturation of 118–125% was measured downstream of the dam.

Several recent disease incidents have occurred at steam-generating facilities. DeMont and Miller (1972) reported mortalities and signs of gas bubble disease in several species of fish at the Marshal Steam Station on Lake Norman, North Carolina, during 1970–1971. The disease occurred among fish in the discharge area during late winter and spring. No dissolved gas measurements were taken in this study, though Adair and Hains (1974) calculated that dissolved gas levels during this mortality period reached a high of 144% of saturation in March. The supersaturation resulted from temperature increase in cooling water at this steam station. Miller (1974) also reported signs of gas bubble disease in fishes at the Marshal Steam Station during 1971–1972, but the incidence was lower than in the previous year and maximum dissolved gas saturation levels of 131% were recorded.

Marcello and Strawn (1972) attempted to culture three fish species in the discharge canal of a steam-electric generating station at Galveston Bay, Texas. They attributed high mortalities of these fish to gas bubble disease.

Marcello and Fairbanks (1975) discussed a mass mortality of Atlantic menhaden (*Brevoortia tyrannus*) at the Pilgrim Nuclear Power Station, Boston, Massachusetts, in April 1973. An estimated 43,000 Atlantic menhaden died in this brief kill. Fish examined showed typical signs of gas bubble disease. Other fish observed near the thermal plume by scuba divers showed no indication of the disease. Total dissolved gas levels were not determined; however, oxygen levels were as high as 142% of saturation.

Gas supersaturation (up to 110%) of alpine streams in Oregon, due to geothermal heating, was reported by Bouck (1976). Fish in the streams showed no evidence of gas bubble disease, but fish in a trout hatchery supplied with such water died from the disease.

These cases demonstrate the widespread occurrence of gas bubble disease and the variety of situations that can cause supersaturation. Each of these factors is discussed separately, in greater detail, below.

Gas Bubble Disease Syndrome

External Signs

Detection of gas bubble disease requires familiarity with the external signs of the disease. These vary from blatantly obvious signs to subtle signs that will be recognized only with careful study.

The first descriptions of the outward signs of the disease were given by Gorham (1901) and Marsh and Gorham (1905). Numerous descriptions of similar external signs followed for other species of fish (Shelford and Allee 1913; Woodbury 1941; Egusa 1959; Renfro 1963; Shirahata 1966; Wood 1968; Beiningen and Ebel 1970; Wyatt and Beiningen 1971; DeMont and Miller 1972; Elling and Ebel 1973; Dawley and Ebel 1975; Dell et al. 1975; Dawley et al. 1976; Nebeker and Brett 1976; Weitkamp 1976). These and many other recent papers describe the signs discussed below.

Exophthalmia or pop eye is a sign commonly associated with gas bubble disease. Marsh and Gorham (1905) found vesicles (gas bubbles or blisters) frequently occurred behind the cornea and in the loose connective tissue of the eye, producing a severe exophthalmia. Failure to observe this obvious sign should not be considered evidence that gas bubble disease does not exist, as exophthalmia may be absent or present in only a few of the fish suffering the disease. Meekin and Turner (1974) reported exophthalmia in less than 5% of dead juvenile chinook salmon suffering gas bubble disease. Exophthalmia also may lead to a false diagnosis of the disease, as it can result from kidney disease, hypoproteinemia, trauma, or parasitism (Stroud et al. 1975; Weitkamp 1976). Dukes and Lawler (1975) described exophthalmia due to infection with lymphocystis virus. Exophthalmia frequently is associated with chronic gas bubble disease, rather than an acute form caused by very high levels of supersaturation.

A much more commonly reported sign of gas bubble disease is bubbles or blisters under the skin, primarily between fin rays. Marsh and Gorham (1905) described vesicles in the fins and on other external surfaces of several marine fishes. The size and location of these bubbles vary with the severity of the disease and the species of fish. In addition to locations in the fins, blisters frequently occur on the head and in the lining of the mouth (Marsh and Gorham 1905; Weitkamp 1974, 1976; Dawley and Ebel 1975; Dell et al. 1975; Dawley et al. 1976; Nebeker and Brett 1976). Photographs of these signs are in reports by Blahm (1974), Ebel and Raymond (1976), Nebeker and Brett (1976), and Weitkamp (1976). These bubbles gradually increase in size as the length of exposure to supersaturation increases (Marsh and Gorham 1905).

According to Meekin and Turner (1974), the cutaneous bubbles were the most common external sign in juvenile chinook salmon that died of the disease in their bioassay studies. These authors and Weitkamp (1976) reported gas-filled bubbles were most common in the caudal fin. Bubbles were less frequent in the paired fins, and only occasional in the anal fin. Bubbles on the head, opercles, jaws, and mouth generally occurred only after the appearance of bubbles in the fins (Weitkamp 1976).

Hemorrhages frequently accompany bubbles in chronic gas bubble disease. Meekin and Turner (1974) observed hemorrhages in about 38% of fish in their bioassays, frequently at the base of the paired fins but seldom at the base of the caudal fin. Stroud et al. (1975) suggested hemorrhage may be a secondary effect of emboli. The hemorrhages usually occur following the appearance of bubbles in the fins, and seem to indicate an advanced stage of the disease, at least in chronic cases.

Bubbles along the lateral line are the first external sign of gas bubble disease to appear in juvenile salmonids (Schiewe and Weber 1976; Weber and Schiewe 1976). These bubbles are small and not easily recognized without experience, which most likely accounts for their absence in most descriptions of the disease. Dawley et al. (1976) reported bubbles along the lateral lines of 50–100% of juvenile chinook salmon and steelheads (*Salmo gairdneri*) exposed to supersaturated water. These bubbles progressively fill the scale pockets of the lateral line system, reducing the ability of the sensory

units to respond to stimuli (Schiewe and Weber 1976). Scattered bubbles (covering less than 15% of the lateral line) may also occur on fish not exposed to supersaturated water (Dawley et al. 1976). This sign should be considered an indication but not necessarily conclusive proof of gas bubble disease unless the lateral line is extensively involved. Bubbles in the lateral line also appear to be lost rapidly in dead fish. Photographs of bubbles in the lateral line system were published by Dawley and Ebel (1975) and by Weber and Schiewe (1976).

The sign most pertinent to recognition of the disease is probably the appearance of bubbles or emboli in gill blood vessels. Dawley et al. (1976) rarely observed emboli in branchial arteries and gill filaments of live fish being held in supersaturated water, but found them prevalent in dead fish. This is an indication that bubbles in the gills may be a cause of death. Wyatt and Beiningen (1971) found that emboli in the gills may be only externally visible signs under acute conditions; gill damage occurred in all fish checked during their acute bioassay tests.

Abnormal behavior is an obvious but rather nonspecific sign exhibited by fish with gas bubble disease (Marsh and Gorham 1905; Bouck et al. 1970; Coutant 1970). Marsh and Gorham (1905) described a loss of equilibrium in fish with the disease. Wyatt and Beiningen (1971) found that in a rapidly lethal or acute situation (about 150% total gas pressure or higher), fish suddenly lost the ability to swim against a current, were unable to avoid obstacles, soon lost equilibrium, moved near the surface without an apparent sense of direction, and then exhibited violent writhing movements interspersed with periods of inactivity. Dawley and Ebel (1975) reported behavioral changes and reduced growth in juvenile chinook salmon exposed to 115% total gas pressure (TGP) in shallow water. Feeding responses became lethargic after 20 days exposure. Many fish developed spinal flexures, exophthalmia, and large blisters in the buccal cavity, and did not accept food. Meekin and Turner (1974) reported northern squawfish (*Ptychocheilus oregonensis*) in supersaturated water failed to feed on juvenile salmonids. In general, the behavior of fish with gas bubble disease appears to follow that expected from any fish suffering severe physical stress that interferes with respiratory and equilibrium functions.

Signs in Eggs

The signs of gas bubble disease vary with the life stages of the fish. In general, eggs appear to be resistant to the effects of supersaturation. Rucker and Kangas (1974) reported no signs of the disease in chinook salmon eggs held in water up to 128% TGP. Meekin and Turner (1974) likewise found no signs of it in chinook salmon eggs but reported heavy mortality of steelhead eggs reared in supersaturated water, although they did not describe gas bubble disease signs in them.

Signs in Larvae and Fry

In larval fish, gas bubble disease appears quite differently than it does in juvenile and adult fish. Herring larvae reared in supersaturated water developed gas bubbles in their guts, which resulted in death (Dannevig and Dannevig 1950; Henly 1952). Similar bubbles developed in flatfish fry but these fish were able to pass bubbles through the anus and survive. Gas bubbles may form on the surface of newly hatched trout fry reared in supersaturated water, causing them to rise to the surface, or bubbles may form in their mouths causing suffocation (Shirahata 1966; Peterson 1971).

Embody (1934), Wood (1968), Rucker and Kangas (1974), and Stroud et al. (1975) described similar signs of gas bubble disease in salmonid fry (sac fry). Bubbles formed between the yolk sac and the perivitelline membrane causing fry to swim head up. As the bubbles expanded and moved posteriorly the fry swam tail up and eventually belly up. Photographs of this condition were included by Harvey and Cooper (1962) and by Shirahata (1966). Death occurred when the vitelline membrane ruptured. Free bubbles occurred in the digestive tracts of all fry, including controls. Adams and Towle (1974) described erratic swimming behavior of coho salmon fry (*Oncorhynchus kisutch*) having large gas bubbles in the yolk sac. No damage to gills or other tissues was found by these authors. Rucker and Kangas (1974) reported that fry that were able to survive bubbles in the yolk sac later developed signs of the disease similar to those described above for juvenile salmonids. Zirges and Curtis (1975) reported large gas bubbles formed in the posterior portion of the yolk sac of chinook salmon sac fry. Dead fish had frayed fins and coagulated yolks, as previously described (Shirahata 1966; Wood 1968). Jones and Lewis

(1976) found bubbles in the peritoneal cavity of channel catfish fry (*Ictalurus punctatus*) with gas bubble disease.

Signs in Adult Salmonids

The external signs of gas bubble disease in adult salmonids are similar to those described for juveniles but generally occur with a different frequency in the various tissues. Adults frequently develop exophthalmia and bubbles in the roof of the mouth near the branchiostegal region (Westgard 1964; Coutant and Genoway 1968; Wood 1968; Beiningen and Ebel 1970). Gas bubbles of varying size may or may not be present in the fins and other exposed surface areas. Nebeker, et al. (1976) described gill damage, hemorrhaging, and bubbles in the oral cavity and on the body surface of adult sockeye salmon (*Oncorhynchus nerka*) exposed to 110–120% TGP in shallow water. Exophthalmia due to the disease may lead to the development of cataracts according to Poston et al. (1973). They found the incidence of cataracts was highest in fish held under conditions most likely to produce supersaturation although they did not measure dissolved gas levels. Cataracts, however, may be due to other causes, as described by Hoffert et al. (1971).

Bouck et al. (1976) and Stroud et al. (1975) also described various signs of gas bubble disease in adult salmonids. Bouck et al. (1976) found adult chinook salmon swam aimlessly, were unresponsive, and exhibited a coughing syndrome as they approached death. These fish also tended to remain at the maximum available depth during the bioassays, thus utilizing the available hydrostatic pressure to compensate for supersaturation. Emboli in the gills caused discoloration of this tissue in dying fish. Stroud and Nebeker (1976) described in greater detail the external signs of gas bubble disease in adult chinook salmon. At 120% TGP, in shallow water, massive blisters occurred in the fins, body surface, mouth, and gills, and behind the eye, as in juvenile salmonids.

Signs in Nonsalmonid Fishes

Bentley et al. (1976) described gas bubble disease signs and lesions in northern squawfish: adults contained gas bubbles between the fin rays and in the subcutaneous layer of large areas of the body surface. Hemorrhages in the subcutaneous layer were also common, but ex-

ophthalmia was rare. Emboli were found in the gill arches of all fish killed in bioassays and in most survivors of 117% and 120% TGP in shallow water. As with adult chinook salmon, northern squawfish remained on the bottom of test containers, thereby minimizing their exposure to supersaturation.

Jensen (1974) described exophthalmia and bubbles in the fins of warmwater species having gas bubble disease. Emboli occurred in the gills of some of the fish showing external signs of the disease. White bass (*Morone chrysops*) from the same area showed a high incidence of fungus infections that may have secondarily invaded the lesions.

The signs and lesions of gas bubble disease in adult Atlantic menhaden were described by Clay et al. (1976), Marcello and Fairbanks (1976), and McLeod (1978). Erratic swimming behavior occurred at 95% TGP (105% N₂). At 107% TGP (110% N₂) mucus secretion, erratic swimming, and color change, along with bubbles in the eyes, intestines, pyloric caeca, and mesenteries, were noted in two fish that died within 96 hours. All fish tested at 110% TGP (120% N₂) died within 24 hours, apparently due to bubbles in the bulbous arteriosus and gill arterioles. These fish also showed the external signs described above.

Signs in Invertebrates

Invertebrates are also susceptible to gas bubble disease. Marsh and Gorham (1905) found it in American lobsters (*Homarus americanus*), horseshoe crabs (*Limulus* sp.), bivalve mollusks, hydroids, and sea spiders (*Anoplodactylus* sp.). The American lobsters and horseshoe crabs survived a long time with their blood in a condition resembling foam. These crustaceans usually lived much longer than fish under the same conditions of supersaturation. Legs of sea spiders became filled with gas and developed a pale color. Mollusks, hydroids, and some green algae developed and emitted bubbles. Hughes (1968) also reported the disease in American lobsters but did not describe any signs.

Malouf et al. (1972) reported gas bubble disease in oysters (*Crassostrea gigas* and *C. virginica*) and clams (*Mercenaria mercenaria*) held in heated water. The oysters first developed crescent-shaped conchiolin blisters in the shelf bordering the mantle. Gas bubbles were also observed in the gill filaments and in the outer layers of

the mantle. In severe cases, the shell cavity became filled with conchiolin blisters. Clams showed obvious lightening of the gill coloration due to the presence of bubbles that prevented free blood circulation. Henly (1952) indicated oyster larvae suffer from the disease but gave no description of it in these molluscs.

Evans and Walder (1969) caused gas bubble disease in shrimp (*Crangon crangon*) by means of decompression. Bubbles in the shrimp were clearly visible through the carapace. Bubbles commonly appeared under the dorsal side of the carapace but were also observed at other locations such as at the base of a leg. Lightner et al. (1974) found the brown shrimp (*Penaeus aztecus*) exhibited erratic and disoriented swimming when affected by gas bubble disease. This behavior was followed by a stupor and helpless floating prior to death. Emboli occurred throughout the tissues of these brown shrimp. Supplee and Lightner (1976) reported it in California brown shrimp (*Penaeus californiensis*) due to oxygen supersaturation. Onset of the disease was marked by erratic swimming behavior. Shrimp then floated near the surface with the ventral side of the head higher than the tail region. Bubbles were apparent in the gill processes and appendages and over the entire body surface; however, few shrimp died.

Nebeker₂ et al. (1976) found that gas bubble disease in daphnia, crayfish, and stone flies caused bubbles throughout their body fluids and tissues, and general body distention prior to death, and they photographed these conditions. Nebeker (1976) reported the daphnia developed massive air bubbles in their gut and under the carapace in the brood pouch. Crayfish were immobilized for many hours prior to death, with signs ranging from nothing observable to massive blockage of body fluids and emboli in the gills. The immobilized crayfish frequently became turgid and swollen from the internal pressure of gas and osmotic imbalance. Stone flies developed bubbles at the base of their legs and gills and in other areas such as the mandibles that were visible through the body wall.

Johnson (1976) described the pathology of gas bubble disease in blue crabs (*Callinectes sapidus*). Dying crabs had hemal spaces of many gill filaments filled with gas. Live crabs had grossly visible gas emboli in hemal sinuses, large arteries, heart, and, occasionally, the midgut lu-

men. Focal degenerative changes occurred in muscle and nervous tissues, due to ischemia. Pathology of the gills was confined to mechanical disruption and displacement of tissues. Additional details of cellular damage are described by Johnson.

Internal Lesions

Internal lesions characteristic of gas bubble disease can be noted by necropsy or histopathological examination. Marsh and Gorham (1905) found diseased fish contained notable quantities of free gas in blood vessels. The amount of gas varied from a few small bubbles to large quantities that distended the heart and caused complete occlusion of vessels. The gas-filled auricle continued beating without propelling blood. The walls of the auricle and ventricle were often emphysematous. Gas bubbles in the gill filaments were described as the most constant and significant lesion. Pathological changes associated with the disease have been discussed in a number of early papers (Renfro 1963; Harvey 1964; Pauley and Nakatani 1967; Bouck et al. 1970; Wyatt and Beiningen 1971). Gas bubbles filled the branchial arterioles causing degeneration of gill filaments, which became edematous, developed aneurysms, and sometimes contained hemolyzed red blood cells.

Pauley and Nakatani (1967) also described degenerative changes in many other tissues of young salmon. Diseased fish had enlarged spleens with hemolyzed red blood cells and a reduction of white pulp. The kidneys, liver, and intestines of those fish showed serious necrotic changes. Degenerative changes in the epithelium of the roof of the mouth were described as unique to gas bubble disease. The extensive tissue changes described by Pauley and Nakatani appear inconsistent with the reported ability of fish to recover from this disease (Marsh and Gorham 1905; Rukavina and Varenika 1956; Shirahata 1966; Weitkamp 1976). It is possible the changes described are indicative of chronic gas bubble disease or some other disease syndrome present in these fish.

Bouck et al. (1970) studied the histopathology of adult sockeye salmon suffering from gas bubble disease downstream from John Day Dam on the Columbia River in 1968. No significant changes were observed that could be attributed to the disease other than those as-

sociated with exophthalmia. Strout et al. (1975) reported emphysema (bubbles in tissue) beneath the peritoneum covering the kidney and ribs, and occasionally under the epicardium in the region of the bulbus arteriosus, in several species of juvenile salmonids. Petechial hemorrhages occurred in muscles, gonads, nares, brain, and other tissues. Edema of the mesenteries was often accompanied by ascites (fluid accumulation in the abdomen). A unique lesion, muscular emphysema, occurred following prolonged exposures (400 hours) to 115% TGP in shallow water. Exophthalmia was due to gas accumulation in the fatty tissue of the orbit. Bubbles in the choroid plexus caused detachment of the retina and rupture of blood vessels. Beyer et al. (1976a, 1976b) reported similar lesions in decompressed juvenile coho salmon. Stroud et al. (1975) were unable to detect the tissue changes reported by Pauley and Nakatani (1967). Stroud and Nebeker (1976) described the internal lesions of gas bubble disease in adult chinook salmon. At 120% TGP, gas-filled spaces were common in the muscle tissue. Blindness, which was common in adult salmonids with the disease, was due to emboli in the choroid plexus causing detachment of the retina and hemorrhaging into the eye. Photographs of internal lesions were included by D'Aoust and Smith (1974), Woelke et al. (1974), Stroud et al. (1975), and Beyer et al. (1976b).

Cause of Death

Although gas bubble disease produces a variety of signs and lesions, the cause of death has generally been attributed to anoxia resulting from stasis of the blood. Marsh and Gorham (1905) found gas to be present in sufficient quantities to completely fill and distend the bulbus of the heart, preventing movement of blood even though the heart continued to beat. Lesser amounts of gas in the vascular system may cause emboli only in the gills (Woodbury 1941; Renfro 1963; and others); these can cause blood stasis in the gill arterioles, leading to death. Dawley et al. (1976) found bubbles in the gills of dead fish but seldom in live fish; they concluded "these signs are directly associated with death."

Stroud et al. (1975) described similar roles of emboli in death caused by gas bubble disease but also mentioned other factors. Sublethal effects such as blindness, stress, and decreased lateral line sensitivity can lead to death from

other causes, such as predation. Gas bubble disease lesions can also increase susceptibility to other diseases. Weitkamp (1976) found fish that were not able to recover from gas bubble disease apparently died due to secondary fungal infection.

Recovery from Gas Bubble Disease

Early in the history of gas bubble disease investigations it was recognized that fish can recover from the disease. Gorham (1901) caused the signs of the disease to disappear in marine fish from the Woods Hole aquarium. He subjected small scup (*Stenostomus chrysops*) showing exophthalmia and emboli of the fins and head to a pressure equal to 4.9 m of water. All signs disappeared within 24 hours. Similar results were found with adult fish. Henly (1952) used hydrostatic pressure to alleviate signs of the disease in young cod (species not given). The fish were held at depths of 2–5 m in natural seawater to affect recovery from the disease.

Although a report on the pathology of gas bubble disease indicated that the fish would not survive the associated tissue damage (Pauley and Nakatani 1967) there have been numerous reports of fish recovering from the disease; several recent ones have provided some insight into why recovery does or does not occur. Tests have used equilibrated water, hydrostatic pressure, and artificially produced pressure to promote recovery.

Meekin and Turner (1974) demonstrated the recovery of juvenile chinook salmon following exposure to 120% and 135% N₂ (110% and 122% TGP). Following exposure times of 4–67 days, the distressed fish were placed in equilibrated water (100% N₂). Seven of the 67 distressed fish died within the first 24 hours. The remaining fish were released in apparently healthy condition after a 2-week period. The sick fish that did not recover included both fish with severe external signs and fish with no external signs of gas bubble disease.

Schiewe (1974) found that recovery of fish from sublethal exposure to supersaturation may occur rapidly. The swimming performance of chinook salmon exposed to 120% TGP showed no effect due to the supersaturation exposure when the fish were allowed to recover in equilibrated water for as little as 2 hours. This rapid recovery corresponds to the rapid disappearance of external signs of gas

bubble disease described by Dawley and Ebel (1975) and Dawley et al. (1976). These authors found that after a recovery period of 2 weeks, most fish no longer showed any external signs of disease.

Weitkamp (1976) tested the recovery of juvenile chinook salmon by holding the sick fish at increased depth to decrease the level of supersaturation through increased hydrostatic pressure. Fish with obvious signs of the disease following exposure to 118–126% TGP for 10 or 20 days were held at a depth of 3–4 m for 20 days. Most of these distressed fish recovered and were released with no apparent signs of gas bubble disease; about 10% died. Most of the fish that died developed fungal infections of the caudal fin, apparently a secondary infection due to lesions in this region. Lesions of the caudal fin were never accompanied by hemorrhages as were lesions of other locations on the fish. The complete lack of hemorrhages at this site indicates the circulation to the caudal fin lesions may not be adequate to prevent overwhelming secondary infections in some cases. The abnormal fungal infections described by Jensen (1974) in largemouth bass (*Micropterus salmoides*) may have also been due to gas bubble disease lesions.

Adams and Towle (1974) used a recompression chamber to alleviate symptoms of the disease. Coho salmon sac fry that showed erratic behavior caused by large bubbles in their yolk sacs were subjected to a pressure of four atmospheres. All of the fish then appeared normal. Decompression was not attempted due to the costs involved. This method does not appear worth serious consideration when similar results have been obtained with only equilibrated water (100% TGP or less) or hydrostatic pressure produced by a head of only 3–4 m.

Supplee and Lightner (1976) were able to reverse gas bubble disease caused by oxygen supersaturation in shrimp. Supersaturation of the water was reduced by vigorously jetting fresh seawater into the surface of raceways. Even the most severely affected shrimp usually recovered within 4–8 hours, and there were few deaths.

Physiological and Behavioral Effects

Marsh and Gorham (1905) were the first to consider the physiological aspects of gas bubble disease. They showed that bubbles forming in supersaturated water contain gases in the same

proportions as those found in air, whereas bubbles in blood are depleted in oxygen. On this basis they concluded that hemoglobin has the capacity to modify the effect of oxygen supersaturation by combining with dissolved oxygen and removing it from the dissolved state. The actual role of this mechanism in gas bubble disease has not been fully investigated; however, several reports discussed in the following section indicate oxygen must reach very high levels to cause the disease if nitrogen levels are low. Beyer et al. (1976b) discussed this relationship in great detail.

Swimming performance following exposure to supersaturation was studied by Schiewe (1974). He found the time of active swimming and the total distance swum by juvenile chinook salmon was affected by exposure to supersaturation. At exposures of 120% TGP in shallow water, swimming performance was significantly lower than in control fish. At 117% and 120% TGP, a significantly greater percentage of exposed fish was unresponsive during the swimming performance test. Schiewe suggested that reduced swimming performance resulting from exposure to supersaturation would make these fish more susceptible to predators. His suggestion assumes that the predators are not likewise affected. Meekin and Turner (1974) and Dawley et al. (1975) found that northern squawfish subjected to supersaturation showed no, or reduced, feeding on juvenile salmonids. Northern squawfish, in their natural habitat, may maintain an adequate average depth to compensate for the levels of supersaturation commonly encountered.

Beyer et al. (1976a, 1976b) demonstrated the rapid equilibration of gas pressures that occurs within fish tissues. In these tests, fish were subjected to internal and external supersaturation and a combination of both. Internal supersaturation was produced by decompressing fish exposed to pressures greater than two atmospheres. External supersaturation was produced by exposure to supersaturated water (150% TGP) at a pressure of one atmosphere. Fish were more sensitive to external than to internal supersaturation. There apparently are slightly different mechanisms related to the direction of the supersaturation gradient and the total volume of gas that account for this difference. Equilibration of the critical tissues occurred in 60–90 minutes at any level of supersaturation. At levels less than 150% TGP,

mortality cannot be directly related to the equilibration time. The mechanism responsible for formation of the emboli that block portions of the circulatory system, thereby causing death, appears to be regulated by some factor or factors other than equilibration time.

These studies also clearly demonstrate the ability of fish to recover from exposure to supersaturation or to avoid supersaturation by sounding. Beyer et al. (1976a) stated that the fish will not eliminate the gas in their tissues when they sound and thus the tissues again will be supersaturated on return to the surface. This may not be a problem for fish sounding frequently in natural waters. As these authors have shown, the time to formation of emboli that apparently cause death or sublethal effects is considerably longer than the 60–90 minutes required for equilibration of the tissues. This may enable a periodically sounding fish to spend a significant portion of each day near the surface without producing the measurable effects of gas bubble disease. This may account for the greatly reduced evidence of the disease in fish intermittently exposed to supersaturation by daily changes in their depth (Meekin and Turner 1974; Weitkamp 1976).

Schiewe and Weber (1976) and Weber and Schiewe (1976) described the sublethal effect of bubbles in the lateral lines of juvenile steelheads. In supersaturated water, bubbles may form along the lateral line in the scale pockets, where they block activity of the afferent lateral line nerve fibers, preventing the lateral line from responding to stimuli. The loss of response, however, was completely reversed after 16–20 hours in equilibrated water. The loss of ability to respond to stimuli decreases a fish's capacity to detect and locate predators or stationary objects. Although this may be an indirect cause of death, as the authors stated, predators may be similarly affected (Meekin and Turner 1974).

Newcomb (1976) described changes in the blood chemistry of juvenile steelheads following sublethal exposure to supersaturation. Increases in blood potassium and phosphate content over that of control fish were found in fish that were subjected to swimming performance tests following exposure to 116% TGP. Concentrations of albumin, calcium, cholesterol, alkaline phosphatase, and total protein in the blood of test fish were decreased. No major changes occurred in the blood chemistry of fish

exposed to 110% TGP or less. The test fish had a 46% incidence of gas bubble disease, primarily characterized by lateral line bubbles. According to Newcomb, the data support the theory that hypoxia rather than starvation occurs in gas bubble disease, although the decline in cholesterol is not explained by this theory.

Changes in hemostatic variables of coho salmon under conditions of decompression were reported by Casillas et al. (1975). Rapid decompression, producing internal supersaturation, caused fibrinogen concentrations to decrease initially, rise within 10–15 minutes, and then decrease to one-half original levels after 1 hour. Partial thromboplastin times increased after 10–15 minutes. Prothrombin times increased almost immediately in juvenile coho salmon and after approximately 1 hour in adults. Decompression apparently activates a hemostatic mechanism that results in consumption coagulopathy.

Casillas et al. (1976a) reported that numbers of thrombocytes (platelets) in the blood of juvenile salmon decreased significantly following decompression. This response was highly dependent on depth and rate of decompression. Thrombocyte numbers returned to normal within 48 hours in most cases. Erythrocyte levels increased significantly 1 day following decompression, which suggests hemoconcentration. Leucocytes showed no significant response; however, conditions of the test may have masked any response. Casillas et al. (1976b) discussed a possible role of the blood clotting mechanism in gas bubble disease. Their discussion is based on increased thrombocyte counts that have been found with physical stress but decreased numbers of thrombocytes were not detected in the decompression experiments.

Bubbles forming in the vascular system and tissues of the fish appear to be the mechanism that causes death, so it is important to understand how they form. There is considerable information available on this subject because bubble formation in the vascular system has been studied extensively in regard to decompression sickness of humans. Marsh and Gorham (1905) attributed the release of gas within the vascular system of fish to a rise in blood temperature (1–7 C) as it passes from the gills to the systemic system. Marsh and Gorham also pointed out the need for gas nuclei for bubble formation. They stated that the blood cells provide loci for

the separation of gas from supersaturated blood. No evidence is offered for this assumption. Rulifson and Abel (1971) briefly described current theories of bubble formation in relation to gas bubble disease.

The role of gas nuclei in bubble formation with regard to decompression sickness has been thoroughly discussed in several papers (Harvey et al. 1944; Harvey 1951; Evans and Walder 1969; Rulifson and Abel 1971). Bubbles will form most easily at an interface of water and another substance where gas nuclei are normally present in supersaturated conditions. Evans and Walder (1969) indicated that these nuclei normally are present in vivo but that they can be destroyed by exposure to high pressure, at least in shrimp. Gas nuclei decrease either the solubility or the surface tension of the supersaturated water, allowing the excess gas to come out of solution. Thus, gas nuclei allow the formation of gas emboli in fish that reside in supersaturated water. Hemmingsen (1970) was able to saturate distilled water with oxygen at 140 atmospheres and with nitrogen at 170 atmospheres without cavitation (bubble formation) when the pressure was released.

Bubble formation in animals is influenced by a number of factors such as muscular activity (Whitaker et al. 1945; Harris₁ et al. 1945). In fact, muscular activity is essential for bubble formation. At very high levels of gas supersaturation even the small muscular movements involved in breathing will cause bubble formation. High concentrations of metabolic carbon dioxide have also been implicated in bubble formation (Harris₂ et al. 1945; Whitaker et al. 1945), but are probably not significantly involved in gas bubble disease problems, as the concentration of this gas does not normally reach supersaturation. It has been suggested that nitrogen may fulfill a similar role if present at supersaturated levels (Berg et al. 1945). Mechanical agitation, such as movement of a bone fractured prior to decompression, also is sufficient to cause bubble formation.

Bubble formation also may be related to the fat content of an animal. Gersh et al. (1944) found the number, size, and location of extra vascular bubbles showed a positive correlation to fat content. Berg et al. (1945) found high fat content per se was not sufficient to increase susceptibility to bubble formation.

Analysis of the gas content of bubbles in fish suffering from gas bubble disease has been re-

ported in several papers (Englehorn 1943; Shirahata 1966). The composition of the gases was essentially the same as the dissolved gas content of water that had been supersaturated with air. The analysis of bubbles described above and by Harvey (1961) has been said to indicate that nitrogen alone is not responsible for gas emboli characteristic of gas bubble disease. It should be noted that the analysis of the bubbles formed is not evidence that the disease is due either to total gas supersaturation, or to nitrogen alone. Harris₂ et al. (1945) showed that bubbles equilibrate within a few seconds to the composition of gases in the surrounding medium. The methods used to analyze emboli have not been capable of measuring the gas composition prior to the rapid equilibration that takes place naturally. Nitrogen could cause the formation of emboli which would rapidly take up oxygen, resulting in bubbles with a gas composition similar to that of the surrounding medium.

Nitrogen-Oxygen-Total Gas Pressure

A question that has received considerable attention in recent years is the role of nitrogen partial pressures versus total dissolved gas pressure (TGP) in causing gas bubble disease in aquatic organisms. Most of the studies reported in the 1960's and early 1970's considered only dissolved nitrogen gas as important in causing the disease. This emphasis apparently was based on the assumption that nitrogen, being biologically inert, was the causative agent while oxygen supersaturation would be regulated or reduced by biological processes.

It has been indicated in several reports that oxygen alone can produce gas bubble disease. Plehn (1924), Woodbury (1941), Alikunhi et al. (1951), Renfro (1963), and Supplee and Lightner (1976) all reported instances of the disease caused by very high concentrations of oxygen. In these cases, oxygen generally reached 300% of saturation or higher, but dissolved nitrogen was not measured. Wiebe and McGavock (1932) were unable to produce gas bubble disease in several species even though oxygen exceeded 300% of saturation. Harvey and Cooper (1962) pointed out the distinction between physical (abiogenic) and biological (biogenic) oxygen supersaturation. Abiogenic oxygen supersaturation occurs with nitrogen supersaturation and the two gases together rapidly induce the disease. Biogenic oxygen produces deleterious ef-

fects only at very high levels near 300% of saturation or higher.

There has been one report that CO₂ supersaturation can cause gas bubble disease (Mrsic 1933). The data presented, however, raise questions as to the validity of the conclusion. As discussed above, Mrsic reported a very high incidence of the disease with a very slight increase in CO₂ from 135 to 138 mg/liter. Other workers (Weigelt 1885; Winterstein 1908; Reuss 1910; Wells 1913; Gutsell 1929) have discussed the toxicity of carbon dioxide to fish but have not indicated that it may be a cause of gas bubble disease.

The conclusion that the disease is probably due to total gas pressure rather than just dissolved nitrogen was indicated by Shelford and Allee (1913) and Doudoroff (1957). It was originally pointed out by Marsh and Gorham (1905) that only two gases, oxygen and nitrogen, are dissolved in water in important quantities. The very small amount of argon dissolved in water is frequently reported together with nitrogen (nitrogen and argon) as both are inert gases. In blood, nitrogen is held in simple solution, while oxygen is primarily bound to hemoglobin. It has not been determined whether hemoglobin does or does not have an effect on the partial pressure of oxygen in simple solution in the blood under conditions of total dissolved gas supersaturation.

Rucker and Kangas (1974) exposed salmonid fry to constant nitrogen partial pressures (120%) at different total dissolved gas pressures. The test fish were exposed to 116% and 120% TGP with each having 120% nitrogen saturation but different oxygen partial pressures. At 120% TGP, the fry showed a significant upsurge of mortality at 25 days while the same upsurge did not occur until 35 days of exposure at 116% TGP. These results indicate that the total gas pressure is more important than the nitrogen partial pressure (N₂) in producing gas bubble disease.

Rucker (1976) also found that various nitrogen and oxygen ratios normally encountered in supersaturated waters do not show a significant difference in their ability to produce the disease. The effect of a constant total gas pressure is significantly reduced only when the oxygen partial pressure is increased considerably in relation to the nitrogen partial pressure. Rucker reported that a drastic decrease in the lethal

effect occurred at 119% TGP when the oxygen and nitrogen partial-pressure ratios were changed from 159%:109% to 173%:105%.

Nebeker₁ et al. (1976) investigated the effects of carbon dioxide and oxygen on the disease-producing ability of a constant total dissolved gas pressure. When carbon dioxide over a range of 2.1–22.0 mg/liter was used, there was no effect on the survival of fish exposed at a constant total dissolved gas pressure. Variation of the oxygen concentration, however, produced differences in survival. At 120% TGP, 50% of the test fish died in 65–70 hours when dissolved oxygen was 11.5–11.8 mg/liter, but only 2% died at 17.2 mg O₂/liter.

Meekin and Turner (1974) exposed juvenile chinook salmon to supersaturated well water in containers 10 cm deep and to supersaturated Columbia River water in containers 61 cm deep. The well water contained 122% N₂ and 112% TGP while the river water contained 124% N₂ and 123% TGP. In the shallower well water a mortality of only 2–5% occurred in 5–10 days. In the deeper river water with nearly the same nitrogen levels but a higher TGP, the fish suffered 92–100% mortality in 5 to 7 days. If nitrogen alone were responsible for gas bubble disease, mortality in the shallower well water should have equalled or exceeded that in the river water.

Dawley and Ebel (1975) also provide indirect evidence of the roles of these gases in gas bubble disease. They divided shallow troughs into upstream and downstream sections with different groups of test fish in each. Fish in both sections experienced nearly identical nitrogen concentrations while oxygen concentrations were 5–10% (of saturation) lower in the downstream sections. The TGP was 112.1% in upstream sections and 110.0% in downstream ones. Gas bubble disease mortality of 50% occurred in the upstream sections within 20 days while no mortality occurred in the downstream sections. This marked difference indicates the role oxygen partial pressures may play in reducing total gas pressure and thus reducing the propensity to produce the disease.

These studies clearly show the importance of total gas pressure as opposed to nitrogen partial pressure in causing gas bubble disease. Consideration of nitrogen partial pressures alone rather than total gas pressure may indicate that a water source is much more dangerous to the

health of aquatic life than it really is. Several of these studies also show that high oxygen partial pressures can reduce the capacity of a given total gas pressure to produce the disease. This effect occurs only when the oxygen rises far above the partial pressures normally experienced in supersaturated waters (160–175%).

Critical Level of Supersaturation

The maximum or critical level of supersaturation has been defined or assumed to be the maximum level of supersaturation that can be permitted to ensure survival and propagation of aquatic biota. A few early studies indicated that 110% nitrogen saturation was a critical level for young salmonids held in shallow water. When supersaturation was first recognized as a problem in the Columbia River system, the critical level of 110% N₂ was adopted as a water quality standard by several northwestern states and the National Academy of Sciences (Water Quality Criteria 1972). The United States Environmental Protection Agency has since perpetuated this critical level as 110% TGP. More recent studies have shown that this value may be only the minimum level of supersaturation that can be safely tolerated by fish confined to shallow water. This does not, however, apply to most natural situations.

Bentley et al. (1976), Meekin and Turner (1974), Weitkamp (1976), and others have shown that a variety of fish, given the opportunity to sound, can survive for extended periods of time in deep water that is supersaturated at a level considerably higher than 110% TGP without a significant incidence of gas bubble disease or death. Fish in most waters that are likely to be supersaturated assume a depth distribution adequate to compensate for supersaturation well above 110% TGP. Johnson and Dawley (1974) and Weitkamp (1976) have shown that an apparent threshold level exists near 120–125% TGP for young salmon that are held in water of several meters depth. Relatively small increases in this range of supersaturation produce a marked increase in the incidence of gas bubble disease and death. Below this level, the incidence of the disease is low and few deaths occur. This is an indication that the present dissolved gas standards are far more restrictive than necessary to protect the fishery resource under natural conditions.

Ebel et al. (1975) have described how early

mortality estimates for the Columbia River system were considerably higher than a more recent estimate based on additional information. Both Ebel (1973) and Bouck (1976) pointed out the difficulties in applying experimentally derived data to the natural situation. The differences between information derived from the laboratory and natural situations should be seriously considered in establishing or revising dissolved gas standards as well as in estimating mortality due to gas bubble disease.

Tolerance to Supersaturation

In order to prevent or eliminate gas bubble disease, it is desirable to know what levels of supersaturation can be tolerated by fish or other aquatic organisms. Frequently the causes of supersaturation are of sufficient economic, social, and political value to make their total removal unacceptable. Hydroelectric projects and steam power plants are far too valuable to be eliminated. It is, therefore, necessary to determine how much supersaturation aquatic organisms can tolerate under various conditions. With this information more effective management and engineering efforts can be made to reduce supersaturation to acceptable levels in those situations where it cannot be eliminated.

Factors that can affect an organism's tolerance to supersaturation include life stage, size, species, and depth distribution. The various major factors that have been studied are reviewed below.

Salmonids at Near-Surface Pressure

Many of the studies conducted to date, in particular most laboratory studies, have exposed fish to supersaturation in water depths of 0.5 m or less. Although these conditions are seldom encountered in natural waters they are pertinent to fish hatchery conditions and provide a practical means for studying many aspects of the problem.

Meekin and Turner (1974) exposed salmonid eggs and young salmonids to supersaturated water in water depths of 20 cm. At 112% TGP, chinook salmon eggs were not affected while eyed steelhead eggs suffered 50% mortality. No signs of gas bubble disease in the eggs were described by Meekin and Turner. Minimal mortalities of juvenile chinook salmon, coho salmon, and steelheads occurred at 103% and 106% TGP in periods of 30–60 days. Mortali-

ties of 8–100% occurred among several groups of juvenile chinook salmon at 114% TGP in 6 days and 64–100% mortality occurred at 124% TGP in 5–7 days. Juvenile chinook salmon in river water of 124% TGP suffered 92–100% mortality in 5–7 days when held within 0.6 m of the surface.

Dawley and Ebel (1975) and Ebel (1973) exposed juvenile chinook salmon and steelheads to supersaturation in 25 cm of water. At 115% TGP, 7% of the chinook salmon and 50% of the steelheads died after 35 days' exposure. At supersaturations of 120% and 125% TGP, chinook salmon experienced a 50% mortality in 27 and 14 hours, respectively, while 50% of the steelheads died in 33 and 114 hours. No gas bubble disease mortalities occurred at levels of 105% and 110% TGP. Dawley et al. (1976) discussed the long-term effects of supersaturation in shallow water. These papers discuss further results of the experiments discussed above. Mortalities in fish held at 110% TGP increased at periods greater than 60 days. At 60 days the mortality in 110% TGP was 15%, but increased to 70% after 125 days' exposure. Disease not related to gas bubble disease was also involved in mortalities after the first 60 days.

Nebeker and Brett (1976) and Nebeker et al. (1979) exposed juvenile sockeye salmon, coho salmon, and steelheads to 110%, 115%, and 120% TGP in water 0.6 m deep. About 5% of the steelheads and none of the salmon suffered mortalities at 110% TGP during the 26–48-day tests. The steelheads suffered a 50% mortality in 21 days and in 2 days at 115% and 120% TGP, respectively. Sockeye salmon suffered a 50% mortality in similar times while coho salmon had only a 10% mortality in 26 days at 115% TGP. At 120%, coho salmon reached 50% mortality in 5½ days.

Weitkamp (1974) exposed wild chinook salmon smolts to Columbia River water of 100–110% TGP during a simulated migration down the lower Snake and lower Columbia rivers. No evidence of gas bubble disease was found in the fish held within 1 m of the surface at total gas pressures not exceeding 110%. The mortalities that occurred in this study were due to secondary fungal infections, following scale loss caused by screening and handling.

In a live-cage study in the Columbia River, Weitkamp (1976) held juvenile chinook salmon within 1 m of the surface for 10- and 20-day

periods. A mortality of 50% was reached in 10 days at total dissolved gas saturations between 118% and 123%. In a third test, when the supersaturation rose to 125% TGP and higher, 50% of the test population died within 2 days. This study showed a dramatic increase in gas bubble disease mortality as total gas pressure increased.

Blahm (1974) and Blahm et al. (1976) described the exposure of juvenile salmonids and other fish to Columbia River water of ambient dissolved gas content in tanks 1 m deep. Total gas pressures varied from 110% to 126%. During 55 days of exposure, the mortalities of chinook salmon, steelheads, and cutthroat trout were 80%, 80%, and 42%, respectively. The chinook salmon and steelheads suffered about 40% mortalities within the first 10 days, with essentially no deaths occurring during the next 30 days' exposure. During the latter 30 days, the supersaturation remained near 118% TGP. Mortalities of all three species increased considerably during the last 5 days of the tests when supersaturation rose to about 123% TGP with a peak of about 127% TGP. This shallow-water bioassay also provides an indication of a marked increase in mortality as TGP rises about 120%.

Bouck et al. (1976) exposed juvenile and adult chinook, coho, and sockeye salmon, steelheads, and rainbow trout to various levels of supersaturation up to 130% mean TGP in water 0.65 m deep. All fish tested tolerated 110% TGP. At 115% TGP and above, the results were somewhat variable even within a given age-group of a single species. Coho salmon parr suffered 50% mortalities following exposures of 77 and 44 hours during two separate tests at a mean TGP of 125%. Above 115% TGP, such variation may be related to peaks of dissolved gas levels reached during the tests rather than the mean total dissolved gas pressures. In over half of the 10- and 14-day tests at 115% TGP, the test fish did not suffer 50% mortality. At 120% TGP there was 50% mortality in 2 to 10 days while at 125% TGP most test populations reached 50% mortality within 2 days in the shallow-water tests.

The above studies indicate that a dramatic change occurs in both the number of deaths and the time to death at approximately 120–125% TGP in shallow water (1 m or less). At gas pressures below this general level, a low in-

cidence of gas bubble disease will be found in juvenile salmonids and deaths will occur at a low rate. Above 120–125% TGP, mortality due to gas bubble disease increases dramatically. This apparent critical level has not been clearly demonstrated but is indicated by these studies. For juvenile salmonids maintaining a deeper distribution, the critical level would be higher.

Hydrostatic Compensation

Marsh and Gorham (1905) recognized that hydrostatic pressure exerted on a fish provides compensation that limited the effects of supersaturation. The total gas pressure, in percentage of saturation, experienced by a fish may be quite different from the level measured and calculated for a fish subjected to near-surface pressure. Each meter of depth exerts additional pressure that increases the solubility of dissolved gases sufficiently to compensate for approximately 10% of saturation. In the range of depths and supersaturations normally of concern, the rule of 10% compensation per meter of water depth is a useful approximation. This means that a total gas pressure of 120% of saturation at the surface is actually only 110% at 1 m and 100% at 2 m, with no change in the volume of gas dissolved or in the partial pressures. Thus, depth is an important factor in determining the tolerance of fish to supersaturation in natural situations.

Several studies have been conducted in deep tanks to evaluate the effect of depth compensation for salmonids in supersaturated water. Ebel (1973) held juvenile chinook salmon in 2.4-m-deep tanks for 60 days. At 118% TGP, insignificant mortality occurred in the deep tanks compared to 100% mortality in 55 hours for fish held in 0.25 m of water. Dawley et al. (1976) reported further results of the same study. At 124% and 127% TGP, juvenile chinook salmon suffered 67% and 97% mortalities, respectively, in the 2.5-m-deep water. In water 0.25 m deep, mortalities in the same range occurred at lower supersaturation levels of 115% and 120% TGP. At 110% TGP in 0.25 m, 15% mortality occurred while only 5% mortality occurred in 2.4-m-deep tanks at 120% TGP.

Several studies have been conducted with flow-through deep tanks and supersaturated Columbia River water. Blahm et al. (1973) held juvenile chinook and coho salmon in tanks 2.5

m and 1 m deep. During the 72-day test period, the supersaturation ranged from 120% to 130%. In the 1.0-m-deep water, mortalities were 98% and 80%, for the two species, respectively. Mortalities of 50% were reached in 50 days for chinook salmon and in 67 days for coho salmon in the 2.5-m-deep water.

Blahm (1974) and Blahm et al. (1976) described further experiments under the same conditions. During 50–55-day tests, juvenile chinook salmon and steelheads suffered 11% and 6% mortalities, respectively, in 2.5-m-deep tanks compared to 80% mortalities for both species in 1-m-deep tanks (120% to 130% TGP). The majority of the deaths occurred near the end of the test when supersaturation rose to between 123% and 127% TGP. Juvenile cutthroat trout held under the same conditions showed far less depth compensation, with a 42% mortality in 1 m and a 27% mortality in 2.5 m.

A number of studies have attempted to simulate more natural conditions by placing live-cages in supersaturated river water. In live-cage studies at Priest Rapids Dam on the Columbia River in 1966, Ebel (1969) reported dissolved nitrogen saturations ranged from 118% to 143%. Juvenile coho salmon were held at depths of 0.5–1.5 m, 2.5–3.0 m, 2.5–3.5 m, and 0–6.0 m for periods of 8–12 days. Fish held below 2.5 m suffered less than 3% mortality in each test. In the 0–6-m cage, 6% and 16% of the fish died while in the surface cage, mortalities were 100% in the first two tests and 20% in the third test. During the third test (August) dissolved nitrogen concentrations were lower, reaching a low of 118%. Complete dissolved nitrogen data are not given by Ebel.

Ebel (1971) conducted similar tests at Ice Harbor Dam on the Snake River in 1970 where dissolved gas levels ranged from about 127% to 132% TGP during the 7-day tests. Juvenile chinook salmon held in a 0–4.5-m-deep cage suffered 45–68% mortality with most survivors showing signs of gas bubble disease. All fish held within 1 m of the surface died during the four tests. Fish held below 3 m suffered no deaths attributable to gas bubble disease.

Meekin and Turner (1974) tested juvenile chinook and coho salmon and steelheads in live-cages at Wells Dam, suspended at 0–0.6-m, 0.9–1.5-m, and 1.5–2.1-m depths in river water at 123% TGP. Nearly all fish held at 0–0.6 m

died within 3 to 7 days during four tests. At 0.9–1.5 m, chinook salmon suffered 4–44%, and steelheads 24% mortality. At the 1.5–2.1-m depth, chinook salmon had 4–16% mortalities while steelheads had 20% mortality in 14 days. Chinook salmon and steelhead juveniles and northern squawfish held between 2.4 and 3.1 m for 14 days in the 123% TGP river water suffered no mortalities and showed no signs of gas bubble disease. Coho salmon juveniles held at Rocky Reach Dam on the Columbia River in water of 125% TGP for 7 days suffered 100% mortality in the 0–0.6-m cage, 19% mortality at 0.9–1.5 m and no mortality at 1.5–2.1 m.

Meekin and Turner also held juveniles of chinook and coho salmon and steelhead in volition cages extending from the surface to 0.6-m, 2.1-m, and 3.1-m depths. Volition cages permit the fish to occupy the depth of their choosing within the confines of the cages. In this test at 126–127% TGP, all fish in the surface cage died in 3 days, while only 4% of the coho salmon and about 60% of the chinook salmon and steelheads died in the 0–2.1-m volition cage during the 30-day test. In 0–3.1-m cages, only 3% of the chinook salmon died, and none of the other two species died, during 21 days.

Weitkamp (1976) held juvenile chinook salmon in supersaturated river water for 10 and 20 days at various specific depths and in volition cages extending from the surface to 1-m, 2-m, 3-m, and 4-m depths. Supersaturation ranged between 118% and 126% TGP. None of these fish died during the 10-day test. During the 20-day test, however, mortalities of 17%, 21%, 1%, and 1%, occurred in populations held at 0–2 m, 0–3 m, 1–2 m, and 2–3 m, respectively. Most of these deaths occurred when the total gas pressure remained near 125%. During a 20-day live-cage bioassay, when total gas pressures remained near or above 125% for the first 11 days, mortality in all cages increased considerably. Chinook salmon held within 2 m of the surface (0–2-m cage) suffered 30% and 61% mortalities while fish permitted access to 1 m greater depth (0–3-m cage) had 1% and 7.5% mortalities. Juvenile chinook salmon held between the surface and 4 m experienced no mortality during these three tests. A few of the fish in the 0–4-m cage had a few bubbles in their fins at the end of the tests.

These reports indicate that the effect of hy-

drostatic compensation due to depth, in both the laboratory and in the field experiments, is as would be predicted by theory. The hydrostatic pressure compensates for about 10% of supersaturation for each 1 m of water depth. The live-cage studies also indicate that given the opportunity, at least under protected conditions, juvenile salmonids will remain deep enough to compensate for total gas pressures of approximately 120–125%. It is necessary to determine accurately the natural depth distribution of fish in supersaturated waters in order to predict their tolerance of supersaturation under natural conditions. This is an important factor in attempting to estimate or predict losses of fish in various situations. The results of laboratory and field bioassay experiments must be interpreted in terms of all discernible natural conditions if they are to provide accurate predictions of what will really happen.

Ebel (1973) made an attempt to estimate the actual gas bubble disease mortalities of juvenile salmonids in the Snake and Columbia rivers. His estimate was based on only a portion of the information now available, but Ebel's discussion points out the complexities involved in evaluating the deleterious effects of supersaturation on naturally migrating populations when the available information comes from limited laboratory tests. The application of experimentally derived information is also discussed by Bouck et al. (1976) who enumerated many of the factors that must be taken into consideration. Many of these factors have not been adequately considered in the formulation of existing dissolved gas standards.

Several recent studies have been conducted to provide information concerning the depth distribution of the migrating juvenile salmonids in the Columbia River system. The depth distribution is important to a determination of the hydrostatic compensation naturally afforded the fish. Smith (1974) found 58% of juvenile chinook salmon and 36% of juvenile steelheads, collected with a fixed gill net, were taken in the upper 4 m of the water column. In this study of a reservoir forebay, Smith also reported 46% of the chinook salmon and 28% of the steelheads were collected above 2 m; 19% of the chinook salmon and 8% of the steelheads were collected above 1 m.

In another study of depth distribution, Weitkamp (1974) collected small numbers of juve-

nile chinook and coho salmon and steelheads in the Columbia and Snake rivers in drifting and fixed gill nets extending to a depth of 5.5 m. Less than 5% of the chinook salmon were collected above 2 m in 1974. About 20% of the coho salmon were collected between the surface and 2 m, and about 10% of the steelheads were collected above 2 m. The depth distribution indicated that a major portion of the steelheads were below the bottom of the 5.5-m net. These fish were collected primarily in the shallower upstream portions of reservoirs.

Blahm (1974) and Blahm et al. (1976) used a depth sounder to determine the depth distribution of migrating juvenile salmonids. An array of 10 transducers described by Marshall (1976) was placed on the bottom of the Columbia River on a gently sloping beach. Approximately 72% of 776 fish detected with this apparatus were between 0.9 and 2.1 m deep. Two beach seine catches containing 37% juvenile chinook salmon in this area "approximately quantified" the species composition, but the depth distribution of the chinook salmon within the total group observed could not be determined.

The above studies do not provide all the information required to evaluate the effects of dissolved gas on the various species. Collection of fish at a specific depth does not indicate these individuals are at this depth for any significant period of time. They may be moving up and down in the water column or they may be remaining at fairly specific depths for long periods. The volition cage experiments reported by Meekin and Turner (1974) and Weitkamp (1976) indicated that all of the fish spent sufficient time at depth to avoid the effects of about 120–125% saturation. This indicates that fish under field conditions are actually experiencing an intermittent exposure to supersaturation through changes in depth. Fish collected near the surface in supersaturated water would show a high incidence of gas bubble disease and death if they remained continuously near the surface.

Table 1 is a summary of bioassay experiments on the tolerance of salmonids to supersaturation. The table includes test depths that are necessary if these results are to be extrapolated to natural river conditions, or when these studies are used to establish or justify dissolved gas standards.

Intermittent Exposure

Intermittent exposure may increase the level of supersaturation fish are able to tolerate because it increases the time over which a specific exposure accumulates. It also provides an opportunity for recovery to occur, particularly if it is accompanied by depth compensation. Intermittent exposure may occur through either changes in the concentrations of dissolved gases or through changes in depth of the fish.

Alternating exposure to spilled or heated waters with periods of little or no exposure to supersaturated water provides an opportunity for fish to reduce internal supersaturation. Although Beyer et al. (1976b) described evidence that critical tissues become saturated within 60–90 minutes, it is unlikely that the tissues would also equilibrate to a reduction of supersaturation in a similar time. Desaturation normally takes much longer than saturation, as evidenced by decompression tables for saturation diving by humans. No good evidence is available for determining the rate of equilibration to reduced supersaturation for fish.

Fish experiencing intermittent exposure by changing their depth will experience very rapid changes in internal saturation. The pressure change will be immediately transmitted to all tissues thus increasing or reducing internal supersaturation according to the changes in depth. Weitkamp (1976) found juvenile chinook salmon selected depths in 0–4-m volition cages sufficient to avoid death of supersaturations of approximately 125% TGP. This study was not designed to reveal any ability of fish to detect and avoid supersaturation, but other studies have indicated fish do not do so.

The effects of intermittent exposure have been examined in experiments with varying levels of supersaturation. Meekin and Turner (1974) alternately exposed juvenile chinook salmon and steelheads to supersaturated and equilibrated water. Using short exposures of 4–16 hours in 17-cm-deep water, they found juveniles can tolerate 122% TGP for periods of 16 hours if they are returned to saturated water (100% TGP) for 8-hour periods. Blahm et al. (1976) alternately exposed fish for 8 hours in supersaturated water of 110–130% N_2 (? TGP) and 16 hours in saturated Columbia River water per 24 hours, as well as by the reverse daily schedule. The time to death of 50% of the test population was closely related to the length

TABLE 1—Summary of dissolved air supersaturation bioassays of salmonid fishes, compiled from literature sources. O₂: oxygen; N₂: nitrogen; TGP: total gas pressure; LE50: lethal exposure to 50% fish.

Species	Supersaturation	Effect observed	Depth (m)	Reference
Rainbow trout fry	Not determined	"Slowly fatal"—day to weeks; gas blisters on head or mucous membranes; "emphysema of the skin"; death with free gas in heart; emboli in gill filaments	0–1.2	Marsh and Gorham 1905
Rainbow trout 12–15 cm	200–300% O ₂ 520–580% O ₂	No deaths in 14 days No deaths, 24-h exposure	Aquaria	Wiebe and McGavock 1932
Brook trout fry	112% TGP or greater	"Badly affected with gas bubble disease"	Hatchery troughs	Emboly 1934
Rainbow trout fry, Cutthroat trout fry	115% N ₂	"Excessive" mortality; bubbles in fins, under skin, in vascular system, in gills, and in kidneys	Hatchery troughs	Rucker and Hodgeboom 1953
Sockeye salmon alevins	108–120% TGP	Gas accumulated rapidly in yolk sac, 20% mortality	Hatchery troughs	Harvey and Cooper 1962
alevins	106–108% TGP	Some signs of gas bubble disease, 2% mortality		
fry	108–120% TGP	Petechial hemorrhages, necrotic areas on fins, exophthalmia		
Rainbow trout swimup fry	<130% N ₂ 153–166% N ₂	No effect 50% mortality	Hatchery troughs, 12 cm	Shirahata 1966
2.4–2.6 cm	<120% N ₂ 148% N ₂	No effect 50% mortality in 5 days		
2.6–2.9 cm	<110% N ₂ 121% N ₂	No effect 50% mortality		
Chinook salmon adults	118% N ₂	Nearly 50% mortality within 10 days	0.6	Coutant and Genoway 1968
Coho salmon juveniles	~140% TGP	Test times 8–12 days 100% mortality 5–70% mortality 3% mortality 18% mortality	0.5–1.5 2.0–3.0 2.5–3.5 0.0–6.0	Ebel 1969
	~120% TGP	10% mortality 3% mortality 0% mortality 6% mortality	0.5–1.5 2.0–3.0 2.5–3.5 0.0–6.0	
Chinook salmon juveniles	127–134% N ₂	7-day tests 100% mortality 100% mortality 34–86% mortality 2–38% mortality 45–68% mortality	0–0.75 0.75–1.0 1.5–2.0 3.0–4.0 0–4.5	Ebel 1971
Coho and chinook salmon, steelhead juveniles	125–130% N ₂	LE50 18 hours	0.2	Ebel et al. 1971

TABLE 1—Continued.

Species	Supersaturation	Effect observed	Depth (m)	Reference
Chinook salmon juveniles	134% N ₂	5–10% mortality, 7.5 hours	0.6	Wyatt and Beiningen 1971
	152% N ₂	100% mortality, 5 hours		
Cutthroat trout	119–136% N ₂	60% mortality, 59 days	1.0	Blahm et al. 1973
		40% mortality, 11 days		
Steelhead	112–130% N ₂	40% mortality, 49 days	1.0	
		27% mortality, 49 days	2.5	
Chinook salmon	112–129% N ₂	80% mortality, 55 days	1.0	
		6% mortality, 55 days	2.5	
Chinook salmon	112–129% N ₂	80% mortality, 55 days	1.0	
		11% mortality, 55 days	2.5	
Cutthroat trout Rainbow trout Chinook salmon Coho salmon	130% N ₂ for 16 hours/day; 100% N ₂ for 8 hours/day	50% mortality, 72 hours		
		50% mortality, 16–70 hours		
		50% mortality, 120 hours		
		50% mortality not reached (192 hours)		
Cutthroat trout Rainbow trout Chinook salmon Coho salmon (all fish juveniles)	130% N ₂ for 8 hours/day; 100% N ₂ for 16 hours/day	50% mortality, 103.5 hours		
		50% mortality not reached (192 hours)		
		50% mortality not reached (192 hours)		
		50% mortality not reached (192 hours)		
Cutthroat trout	131–139% TGP 125–131% TGP 110–127% TGP 113–122% TGP 102–128% TGP	100% mortality, 3.8 days	0.6	May 1973
		100% mortality, 6 days		
		50% mortality, 2.2 days		
		50% mortality, 14 days		
		No mortality, 12 days		
Mountain whitefish	131–139% TGP 116–127% TGP 113–122% TGP 107–128% TGP	No mortality, 12 days		
		25% signs of gas bubble disease		
		100% mortality, 1.5 days		
		50% mortality, 12 days		
Cutthroat trout	131–139% TGP	40% mortality, 17 days	3.0	
		1 mortality, 17 days		
		75% signs of gas bubble disease		
		50% mortality, 17 days		
Mountain whitefish		55% mortality, 24 days		
		67% mortality, 24 days		
Chinook salmon juveniles	122% TGP 114% TGP 112% TGP 106% TGP	50% mortality, 18 days	0.2	Meekin and Turner 1974
		67% mortality, 24 days		
		32–100% mortality in 3–8 days		
		8–100% mortality in 6 days		
Coho salmon juveniles	112% TGP 106% TGP	8–75% mortality in 18–67 days		
		0–8% mortality in 18–67 days		
		60–100% mortality in 6–35 days		
		0–4% mortality in 28–36 days		
Steelhead juveniles	122% TGP 112% TGP 106% TGP	no signs of gas bubble disease		
		100% mortality in 3 days		
		6–30% mortality in 6–30 days		
		No effect		

TABLE 1—Continued.

Species	Supersaturation	Effect observed	Depth (m)	Reference
Chinook salmon juveniles	123–125% TGP	92–100% mortality in 3–7 days	0–0.6	
		4–40% mortality in 14 days	0.9–1.5	
		4–16% mortality, 14 days	1.5–2.1	
		No effect	2.4–3.0	
Chinook salmon fry exposure from hatching to 50 days old	128% TGP 124% TGP 120% TGP 116% TGP 112% TGP	83% mortality	~0.2	Rucker and Kangas 1974
		75% mortality		
		68% mortality		
		16% mortality		
		12% mortality		
Chinook salmon juveniles	125% N ₂ 120% N ₂ 115% N ₂ 110% N ₂	LE50 13.6 days	0.25	Dawley and Ebel 1975
		LE50 26.9 days		
		LE50 not reached		
		Same as controls		
Steelhead juveniles	125% N ₂ 120% N ₂ 115% N ₂ 110% N ₂	LE50 14.2 days		
		LE50 33.3 days		
		LE50 486 days		
		Same as controls		
Chinook salmon, steelhead juveniles	128% N ₂ 125% N ₂ 100–120% N ₂	28-day test	2.4	
		50% mortality		
		24% mortality No significant mortality		
Cutthroat trout	112–136% N ₂	32–50% mortality	1.0	Blahm et al. 1976
		37–50% mortality	2.5	
Steelhead	112–129% N ₂	70% mortality	1.0	
		0% mortality	2.5	
Chinook salmon (all juvenile fish)	112–129% N ₂	80% mortality	1.0	
		11% mortality	2.5	
		(all corrected for control mortality)		
Chinook salmon adult	130% TGP	8.5–10 hours	<1.0	Bouck et al. 1976
Rainbow trout parr	125% TGP	27–35 hours		
yearling		31 hours		
Sockeye salmon parr		40 hours		
Coho salmon parr		12 hours		
adult		19–21 hours		
Chinook salmon parr		18 hours		
Rainbow trout parr	120% TGP	51 hours		
adult		79–92 hours		
Coho salmon adult		45–51 hours		
Chinook salmon adult		51 hours		
Chinook salmon 3–5 cm	120% TGP 115% TGP 110% TGP 105% TGP 127% TGP 124% TGP 120% TGP 115% TGP 110% TGP	Mortality in 60-day exposure		Dawley et al. 1976
		97%	0.25	
		80%		
		15%		
		<5%		
		80%	2.5	
		65%		
		<5%		
		<5%		
		<5%		

TABLE 1—Continued.

Species	Supersaturation	Effect observed	Depth (m)	Reference	
Steelhead 16.5–19.5 cm		Mortality in 7-day exposure			
	120% TGP	100% (2 days)	0.25		
	115% TGP	57%			
	110% TGP	<5%			
	127% TGP	25%	2.5		
	120% TGP	5%			
	115% TGP	<5%			
Chinook salmon juvenile	119–123%	Mortality in 10-day exposure		Weitkamp 1976	
		53%	0.1		
		0%	0–2, 3, 4		
	120–128% TGP	Mortality in 20-day exposure			
		88–100%	0–1		
		17–61%	0–2		
		3–7.5%	0–3		
		0	0–4		
		1–30%	1–2		
		1%	2–3		
		12–70%	16 hours at 0–1 8 hours at 3–4		
		4–39%	12 hours at 0–1 12 hours at 3–4		
1–7%	8 hours at 0–1 16 hours at 3–4				

of the exposure to the supersaturated water. Less than 50% of chinook and coho salmon, steelheads, rainbow trout, mountain whitefish, and largemouth bass were killed by an 8-hour exposure per day to supersaturated water (130% N₂). Most of these fish, however, suffered 50% mortality in less than 24 hours during continuous exposure to supersaturation (130% N₂).

Controlled changes of depth also have been used to study the effects of intermittent exposure. Weitkamp (1976) intermittently exposed juvenile chinook salmon to Columbia River water by changing the depth of 1-m-deep live-cages on 8-16-, 12-12-, and 16-8-hour schedules. The live-cages were alternated between depths of 1–2 m and 3–4 m for one 10-day test and between 0–1 m and 3–4 m for two 20-day tests in an attempt to represent possible diel changes in migrating fish. No deaths or signs

of gas bubble disease occurred in the 10-day test with saturations between 118% and 123% TGP. At 120–126% TGP, mortalities reached 1%, 4%, and 12% in 20 days of 8-, 12-, and 16-hour surface exposures, respectively. During the second 20-day test, supersaturation rose to near or above 125% TGP for an 11-day period. During this time, 50% mortality was reached in 5 days with the 16-hour surface exposure. In less than 48 hours during the same period, 50% mortality occurred for fish given continuous exposure at 0–1 m. At this higher level of supersaturation (125% TGP), mortalities in the 12- and 8-hour exposure cages were 39% and 7%, respectively. Dissolved gas levels below 125% during the last 9 days apparently enabled the surviving fish to lose all signs of gas bubble disease.

These studies indicate that intermittent exposure either by changes in the supersaturation

of the ambient water or by changes in hydrostatic pressure will allow fish to tolerate supersaturation for a longer period. This increase in the exposure time that is tolerated is greater than the sum of the intermittent exposures, which indicates some recovery occurs during short periods of reduced supersaturation.

Detection and Avoidance

The ability of fish to compensate for supersaturation may be increased if the fish are able to detect and avoid supersaturation. Fish can avoid supersaturation by either refusing to enter supersaturated water when a choice exists or by sounding to compensate for supersaturation at surface pressures.

It has been generally accepted that fish are not able to detect supersaturation and avoid it. Several recent reports indicate that this theory may not be valid for all conditions. This question is of considerable importance as it can greatly affect the extrapolation of experimental data to the conditions faced by fish in natural waters. Fish in natural waters frequently have the opportunity to seek hydrostatic compensation or to avoid entering supersaturated waters if they are able to detect supersaturation.

Ebel (1971) found that juvenile chinook salmon held in 0–4.5-m volition cages suffered much higher mortality from gas bubble disease than fish forced to remain in deeper water (3–4 m). This suggests that these fish were unable to detect, or were unwilling to avoid, supersaturation.

The ability of juvenile chinook and coho salmon to detect and to avoid supersaturation when permitted an alternative in shallow water was studied by Meekin and Turner (1974). A divided trough with supersaturated water at 110–117% TGP on one side and equilibrated water at 101% TGP on the other side was used to test the fishes' response. The fish were introduced to the trough in the lower mixing zone of 110–113% TGP. Juvenile chinook salmon showed a strong preference for the equilibrated water and avoided the supersaturated water when the water supply was switched from one side of the trough to the other. Coho salmon showed no preference for either equilibrated or supersaturated water. These results are not definitive due to temperature differences between the two water sources used during part of the tests. The report does not indicate which

fish were tested with water sources having different temperatures. Differences in the chemical composition of the water also may have influenced the results of these tests, as the two levels of supersaturation were drawn from different supplies.

Blahm et al. (1976) reported tests of juvenile chinook salmon and steelheads in a divided trough having 130% N₂ (? TGP) on one side and 102% N₂ on the other side. Steelheads did not avoid the supersaturation, for they reached 50% mortality in about 43 hours. Chinook salmon apparently avoided the supersaturation for they suffered no deaths during either of the 8-day tests. The results agree with Meekin and Turner's (1974) report of avoidance of supersaturation by juvenile chinook salmon.

Dawley et al. (1976) reported the apparent detection and avoidance of supersaturation in deep (2.4 m) tanks. The vertical distribution of juvenile chinook salmon and steelheads was apparently altered after 3 days' exposure to various levels of supersaturation. For the first days of exposure the depth distributions of various test groups were not significantly different. After 3 days, the mean depth of the groups in the supersaturated water was greater than that of fish in saturated water, and mean depths increased with increasing levels of supersaturation.

Bentley et al. (1976) described the apparent avoidance of supersaturation by northern squawfish below Little Goose Dam on the Snake River. Catches of northern squawfish below the dam were much lower during the period of higher supersaturation than they were before and after this period. The fish apparently either avoided the area of supersaturation or assumed a deeper vertical distribution during the high supersaturation period. Large numbers of northern squawfish were captured in a side arm of the reservoir below Little Goose Dam during this time. These fish may have been avoiding higher dissolved gas concentrations in the main river, although gas pressures in the side arm were not measured.

Stickney (1968) reported Atlantic herring (*Clupea harengus harengus*) showed a definite tendency to avoid supersaturation. This avoidance occurred only when the supersaturation was high enough to produce gas bubble disease: 120% N₂ and 130% O₂ (122% TGP).

These studies indicate some fish may be able

to detect and avoid supersaturation and others may be unable to detect or do not avoid supersaturation. Reported mortalities caused by supersaturation in thermal discharges at electric generating stations indicate that some species are either not able to detect supersaturation or that their attraction to heated water overcomes their aversion to supersaturation. It is obvious that insufficient information is available to draw any useful conclusions on this issue.

Life Stage

The tolerance of fish species to dissolved gas supersaturation is not the same at all life stages. As discussed above, eggs show no signs of gas bubble disease when held in supersaturated water. They appear to be tolerant of levels of supersaturation that affect fish. Marsh and Gorham (1905) and other more recent reports indicate eggs do not develop the disease. Meekin and Turner (1974) provide the single known report of gas bubble disease in eggs. Steelhead eggs developed high mortalities but chinook salmon eggs did not when both were hatched in water having 112% TGP. It is possible that something other than supersaturation was responsible for the steelhead egg mortalities. No discussion of the egg pathology is provided. The majority of evidence indicates fish eggs are extremely tolerant of supersaturation.

In general, the tolerance of different life stages appears to follow two consecutive trends. In very early life stages, the tolerance to supersaturation decreases from very great tolerance in the egg to very low tolerance in older juveniles. Life stages following the juvenile stage appear to increase in tolerance to supersaturation, with adults being generally the most tolerant free-swimming life stage.

Marsh and Gorham, however, reported Atlantic cod (*Gadus morhua*) fry to be tolerant of levels of supersaturation that produced gas bubble disease signs in adult fish; however, they only held the fry in this supersaturated water for 2 days. Egusa (1959) found killifish (*Oryzias latipes*) fry to be resistant to supersaturation immediately after hatching, apparently due to "elasticity and tenacity of tissues of the body wall." Shirahata (1966) reported rainbow trout fry became increasingly less tolerant of supersaturation with increasing age for the first 2 months after hatching.

Meekin and Turner (1974) also reported de-

creasing tolerance with increasing age in juvenile chinook salmon. Mortalities of 100-mm chinook salmon were three to four times greater than those of fish under 40 mm. Larger individuals of coho salmon and steelhead also showed a reduced tolerance to supersaturation. Rucker (1975) compared small (38 mm and 46 mm) coho salmon to larger (100 mm) juveniles in 0.2-m-deep water at 112% TGP. The time to 50% mortality was 2.6 and 4.2 days for two groups of the larger fish, 2.7 days for the 46-mm fish and more than 30 days for the 38-mm fish. Dawley et al. (1976) tested several sizes of juvenile chinook salmon in 0.25-m-deep water at 112% TGP. They found fish 40 mm long to be significantly more tolerant of supersaturation than fish 53 mm and 67 mm long. The smaller fish suffered less than 10% mortality in 45 days while the larger fish reached over 50% mortality in less than 15 days. These studies indicate a general decrease in tolerance of juvenile salmonids with increasing age and size.

On the other hand, some workers have indicated that older fish are more tolerant of supersaturation than young fish. Harvey and Cooper (1962) reported sockeye salmon alevins (sac fry) appear to be particularly susceptible to supersaturation. Wood (1968) described the levels of nitrogen supersaturation detrimental to various life stages of salmonids as 103–104% for fry, 105–112% for young juveniles, and 118% for adults. No indication is given by Wood as to how this information was derived. Bouck et al. (1976) reported the results of a number of bioassays in 1-m-deep water using several life stages of salmonids and other fishes. Although there was considerable variation between individual tests, they found younger fish were generally less tolerant of supersaturation than older fish. At 115% TGP, the mean times to 20% mortality for salmonids were 125 hours for juveniles, 154 hours for smolts, and 309 hours for adults.

Individual fish at any particular life stage have shown considerable differences in their tolerance to supersaturation, as indicated by most of the bioassays discussed above. Becker (1973) discussed a more direct method of measuring individual tolerances than differences in the time to death. The formation of emboli in circulating blood was recorded by a telemetering flowmeter surgically implanted on the conus arteriosus of rainbow trout. The results in-

icated individual fish of this species varied widely in their tolerance to bubble formation in water of the same level of supersaturation.

Heritability

We have found only one study that addressed the heritability of tolerance to gas bubble disease. Cramer and McIntyre (1975) studied the tolerance to supersaturation of several stocks of chinook salmon from Oregon coastal streams and from the lower and upper Columbia River. Stocks with the longest history of exposure to supersaturation were the most tolerant of supersaturation. A comparison of 80 tank families produced from twenty males and four females indicated the tolerance is inherited.

Cramer and McIntyre (1975) made estimates of increases in survival that could be expected with each generation experiencing a given mortality due to gas bubble disease. For mortalities of 30%, 50%, and 70% in the salmon population, increases in survival of 0.4%, 0.7%, and 1.0% should occur with each succeeding generation. These increases are not great but do indicate an advantage to using tolerant stocks in waters that may be supersaturated. This study suggests that experiments on fish from rarely supersaturated waters may indicate more serious problems than are actually encountered by populations in frequently supersaturated waters.

Temperature

As water temperature affects many activities of fish, it is important to determine its relationship to gas bubble disease. The consideration of temperature effects is particularly important in the Columbia and Snake rivers where average water temperatures have been raised by many hydroelectric projects (Beiningen and Ebel 1970). The effect of temperature on tolerance to supersaturation is also important in discharges of heated water that may have become supersaturated within thermal power plants.

A number of studies have been conducted to determine if a synergistic or additive effect exists between high temperatures and supersaturation. Coutant and Genoway (1968) found that chinook salmon acclimated in or tested in supersaturated water (greater than 118% N_2 saturation) could not survive a temperature of 22 C. Fish acclimated and tested in water hav-

ing a lower dissolved gas content (less than 110% N_2) also died, but at a slower rate. Using adult sockeye salmon, Bouck et al. (1970) found temperature increases in saturated water following exposure to supersaturation caused an increased rate of blindness; deaths at higher temperatures (20 and 22.5 C) were related to pathogenic bacteria.

The relationship between temperature and supersaturation for juvenile chinook salmon was studied by Coutant (1970), but the results were inconclusive. Ebel et al. (1971) reported that a stress of 115–120% N_2 for 12 hours did not greatly affect the temperature tolerance of juvenile chinook salmon. When tested in heated water supersaturated at 125–130% N_2 , the prior stress significantly decreased the temperature tolerance of these fish. Fish tested at elevated temperatures in saturated water showed no effect that could be attributed to the prior stress of supersaturation.

The National Academy of Sciences/National Academy of Engineering (1972) concluded that supersaturation has no real effect on thermal tolerance. Becker (1973), however, reported that higher losses of juvenile salmonids occurred among those that had been subjected to the stress of supersaturation than among those that had not. He concluded that the exposure of salmonids to the Hanford thermal plume in the Columbia River was too brief to cause mortalities.

Fickeisen et al. (1976) studied the tolerance of black bullheads (*Ictalurus melas*) to supersaturation at temperatures of 8, 12, 16, and 20 C. Temperature effects were very slight within this range, and not of ecological significance. The TL50's were 126.7% TGP at 8 C and 124.4% TGP at 20 C.

Bouck et al. (1976) found adult sockeye salmon were considerably more tolerant to 120% and 125% supersaturation when they were acclimated slowly from 10 C up to 18 C than when they were permitted little or no acclimation. Young fish showed a variable response; increased temperatures increased tolerance in one test but decreased it in two others. The effect of temperature on tolerance to supersaturation appears to be so slight that it is often overshadowed by other factors. In the tests reported by Bouck et al. (1976), the acclimation temperature and period of acclimation appeared to significantly affect the test results.

Temperature appears to have little direct influence on the tolerance of fish to supersaturation but it does produce an indirect effect by changing the solubilities of dissolved gases. As water temperatures increase the solubility of dissolved gases decreases, thus resulting in greater levels of supersaturation even though the dissolved gas concentration remains constant. This has long been recognized in research on gas bubble disease. Engelhorn (1943) used a rise in water temperature to bring about supersaturation and produce symptoms of gas bubble disease. Supersaturation as a result of warming hatchery water was reported by Rucker and Hodgeboom (1953). Westgard (1964) noted this mechanism was also responsible for a minor portion of the supersaturation problem at the McNary spawning channel. DeMont and Miller (1972), Adair and Hains (1974), Miller (1974), and Marcello and Fairbanks (1976) all discussed aspects of supersaturation associated with thermal effluents.

Malouf et al. (1972) described supersaturation produced by heating cold seawater in closed heat exchangers. The supersaturation was sufficient to cause gas bubble disease in three species of bivalve molluscs. Lightner et al. (1974) described the disease in shrimp resulting from supersaturation produced by heating seawater in a closed system. Zirges and Curtis (1975) encountered similar problems after heating a water supply for chinook salmon sac fry. The various aspects of these reports are discussed in greater detail in other sections of this review.

Isaacson (1977) questioned the role of gas bubble disease in heated effluents. This author pointed out that pop eye, a sign of this disease, can be caused by cold stress which could be confused with gas bubble disease in heated effluents. Although this may be true in some cases, it is unlikely that cold stress would cause emboli and other recorded signs that appear to be specific to the disease.

Saltwater Adaptation

In recent years there has been considerable concern about the ability of juvenile salmonids to adapt to salt water following exposure to supersaturation during their downstream migration. As gas bubble disease produces various tissue changes, described above, it has been theorized that it might also significantly reduce

the ability of the juvenile salmonids to undergo the physiological changes required to make the transfer from fresh water to salt water.

Dawley et al. (1976) described an experiment with chinook salmon and steelheads that had survived supersaturation bioassays. Surviving fish exposed to 110%, 115%, and 120% TGP for 127 days were transferred to 25‰ seawater and held for 13 days. Most of the 50 chinook salmon died in this test; only eight of the larger fish survived the full test period. The majority of the fish may not have reached smolting size and thus did not have the ability to adapt to salt water. Survival was higher among the steelheads tested; most deaths were of smaller fish. The authors concluded that prior exposure to supersaturation seemed not to affect the ability of steelheads to adapt to salt water and that their data on chinook salmon were inconclusive with regard to saltwater adaptation.

Bouck et al. (1976) also tested the ability of various salmonids to adapt to salt water following exposure to supersaturation. Following exposure to 110%, 115%, and 120% TGP, steelhead and sockeye and chinook salmon juveniles were transferred to gas-equilibrated seawater. In all tests the transferred fish either survived for over 5 days, at which time the experiment was ended, or died from causes unrelated to supersaturation. Bouck et al. concluded that no latent or delayed mortalities occur due to gas bubble disease after salmonid smolts enter seawater.

Nonsalmonids

Although much of the recent research and publicity on gas bubble disease has concentrated on salmonids because of the Columbia River problem, there has been considerable research on other species. Gorham (1901) originally described the disease from scup, a marine species. Scup were killed by gas bubble disease when held in shallow aquaria containing water with 135–145% TGP (based on the solubilities used by Gorham). Marsh and Gorham (1905) reported the disease in a variety of other species, presumably at similar dissolved gas levels, but few quantitative details are provided.

Woodbury (1941) observed gas bubble disease in a variety of freshwater fish as the result of oxygen supersaturation caused by photosynthetic activity. Black crappies (*Pomoxis nigromaculatus*), bluegills (*Lepomis macrochirus*), northern

pike (*Esox lucius*), and carp (*Cyprinus carpio*) all died at very high oxygen levels (30–32 ppm).

Dannevig and Dannevig (1950) and Henly (1952) discussed gas bubble disease in artificially hatched Atlantic cod, herring, and plaice (*Pleuronectes microcephalus*), but gave no details about dissolved gas concentrations. Stickney (1968) produced the disease in herring held in aquaria at 122% TGP.

Egusa (1959) compared the tolerance of five species to dissolved gas supersaturation by determining the "detrimental nitrogen limit" (signs of gas bubble disease in 50% of test fish within 2 weeks) and the "lethal nitrogen limit" (50% mortality within 2 weeks). The detrimental limits for the fish were 125% for adult goldfish and eel (*Anguilla japonica*); 130% for young goldfish, young carp, adult killifish, and adult bitterling (*Rhodeus acellatus*). The lethal limits were 120% for adult carp; 125% for adult goldfish; 130% for eel, young carp, bitterling, and young goldfish; over 140% for killifish. Supersaturation values given by Egusa are percent nitrogen saturation, and would be about 5% of saturation lower if reported as TGP.

Renfro (1963) reported gas bubble disease mortality for a number of marine fishes in Galveston Bay caused by oxygen saturations apparently over 250%: spotted seatrout (*Cynoscion nebulosus*); gulf menhaden (*Brevoortia patronus*); bay anchovies (*Anchoa mitchilli*); juvenile Atlantic croakers (*Micropogon undulatus*); speckled worm eels (*Myrophis punctatus*); longnose gar (*Lepisosteus osseus*).

Marcello and Fairbanks (1976) described a mortality of Atlantic menhaden due to gas bubble disease. Supersaturation resulted from temperature increases in the cooling water discharged from the Boston Edison Company's Pilgrim Nuclear Power Station. Dissolved oxygen levels measured during the mortality were frequently between 130% and 140% of saturation. Total dissolved gas levels were likely in the same range or higher. Several other species of fish and invertebrates were observed in the supersaturated area with no evidence of gas bubble disease. Clay et al. (1976) found Atlantic menhaden held in shallow tanks showed signs of the disease at 107% TGP within 96 hours.

DeMont and Miller (1972) and Miller (1974) described the occurrence of the disease in a number of species from Lake Norman, North

Carolina. Signs occurred primarily in white bass, redbreast sunfish (*Lepomis auritus*), bluegills, and threadfin shad (*Dorosoma petenense*). Signs were also reported in a few individuals of ten other species. These fish experienced supersaturation as high as 130% TGP that was produced by the heating of lake water at the Marshal Steam Generating Station.

Fickeisen et al. (1976) studied the tolerance of black bullheads to supersaturation at several temperatures. The dissolved gas level required to produce a 50% mortality of the test population during 96-hour tests (TL50) was about 125% with slight differences depending on the water temperature.

Supersaturation bioassays were conducted by Blahm et al. (1976) on a variety of species, including largemouth bass and mountain whitefish, which were also tested in comparable intermittent-exposure tests. The largemouth bass were more tolerant of supersaturation than the salmonid species while the tolerance of mountain whitefish was about equal to that of coho salmon and steelheads. This conclusion was based on the time to mortality of 50% of the test population (LE50) at 130% N₂ for 24-, 16-, and 8-hour/day exposures. Bioassays of smelt, crappies, and northern squawfish were also conducted by Blahm et al. in river water at ambient supersaturation ranging from about 113% to 123% TGP. Although the tests measured different end points, they did indicate that the tolerance of smelt was similar to that of steelheads but less than that of most salmonids. The crappies and northern squawfish were more tolerant than the salmonids and suffered no deaths in 20 days and 35 days, respectively.

Bouck et al. (1976) reported less than 10% mortality of largemouth bass exposed for 20 days to about 125% TGP in 0.65 m of water. The largemouth bass were able to capture and eat juvenile salmon during this test. Apparently, supersaturation in the ranges normally experienced would have little effect on predation by this species. Bouck et al. (1976) reported shiners (*Notropis* sp.) and crappies (*Pomoxis* sp.) have a tolerance comparable to that of salmonids. Bluegills, northern squawfish, and warmouth (*Lepomis gulosus*) were more tolerant to supersaturation than shiners and crappies but were less tolerant than largemouth bass, bullheads (*Ictalurus* sp.), and carp.

A number of studies have examined the tolerance of the northern squawfish, a major predator of juvenile salmonids during their downstream migration. Meekin and Turner (1974) found adult northern squawfish to be more tolerant to 111% TGP than steelhead and chinook salmon juveniles, but the adult squawfish were of equal or lower tolerance to 122% TGP when tested in shallow water (20 cm). Juvenile chinook salmon and steelheads placed in a trough with northern squawfish were vulnerable to predation at 100% TGP but were more active than the predators, and were ignored by them, at 111% TGP. This indicates that sublethal supersaturation may produce greater effects on a principal predator of salmonids than on the prey.

Bioassays of northern squawfish in constantly supersaturated water were conducted in shallow tanks (0.25 m) by Bentley et al. (1976). Twelve-day mortalities were 100% at 126% TGP; 60% at 120% TGP; 32% at 117% TGP; and 0% at 110, 107, and 100% TGP. At 126% TGP, all fish died in less than 1 day. All survivors of the 120% and 117% TGP tests, and most of the fish exposed to 110% TGP, showed signs of gas bubble disease at the end of the 12 days.

Bentley et al. (1976) also found that northern squawfish collected from the Snake River during periods of moderate to high supersaturation showed less evidence of feeding than the fish collected during times when little or no supersaturation was present in the area. Feeding was decreased by about 50% at 115% TGP. In the bioassays, northern squawfish tended to remain on the bottom of the test tanks during exposure to supersaturation. This behavior may be responsible for the reduced feeding observed in nature, and may contribute to better survival through depth compensation.

Parametrix (1974) sampled resident species downstream from Grand Coulee Dam in Lake Rufus Woods, where levels of supersaturation have been as high as 145% TGP in previous years. During the time of this particular survey, supersaturation reached 110% TGP for a brief period. The presence of a variety of adult fish of several age-classes in Lake Rufus Woods was taken as an indication that the populations were surviving the previous years' high levels of supersaturation. Although recruitment of fish from downstream reservoirs or from tributaries

is not possible in this area it is probable that recruitment of at least some adult fish occurred from Lake Roosevelt, which is behind Grand Coulee Dam. Lake Roosevelt had also been supersaturated (140% TGP) in the previous year (Seattle Marine Laboratories 1972b).

The studies discussed above indicate that the tolerance of different species to supersaturation can vary considerably. Salmonids are among the least tolerant fish but others, such as Atlantic menhaden, may be even less tolerant. In natural situations the tolerance of a species will be affected by behavioral patterns such as depth distribution or attraction to heated waters.

Causes of Supersaturation

Water may become supersaturated with atmospheric gases through any one of several different processes, either caused by humans or nature. These processes either cause an increase in the amount of air dissolved or they reduce the amount of air water will hold.

Lindroth (1957) discussed four ways by which water may become supersaturated: (1) water contains dissolved gas coming from a gas mixture containing a higher percentage of that gas than is normally found in air; (2) water contains gas that was dissolved under a higher-than-atmospheric pressure; (3) water contains gas dissolved at a lower-than-ambient temperature; (4) two bodies of saturated water at different temperatures are mixed.

The first mechanism is probably of little importance as it is likely to be encountered only in experimental situations. The second mechanism has been involved in many of the documented supersaturation problems, indicated in the following discussions of air injection and hydroelectric projects. The second mechanism is also involved in the supersaturation of natural springs. The third mechanism has caused supersaturation in the heating of water supplies for fish culture, the cooling waters of power generating facilities, and in geothermal heating of natural waters. The fourth mechanism may cause supersaturation but is unlikely to produce levels high enough to cause gas bubble disease under most circumstances. An additional mechanism not mentioned by Lindroth (1957) is photosynthesis. Photosynthetic activity has been responsible for several reported cases of the disease.

Air Injection

Any situation that allows air to be mixed with water under pressure much greater than one atmosphere can produce supersaturation if adequate volumes of air are available. The initial description of gas bubble disease and its cause by Marsh and Gorham (1905) resulted from this mechanism. A leak on the suction side of the saltwater supply system for the Woods Hole aquarium system permitted air to be drawn in with the water.

The supersaturation problem at Flodevigen marine fish hatchery was reported by Dannevig and Dannevig (1950). In this instance, air was sucked into the system at the shaft bearings of the pump. Harvey and Smith (1961) described a Canadian hatchery intake system that permitted air to be sucked into the intake under various conditions causing supersaturation. Wyatt and Beiningen (1969, 1971) found a similar situation at an Oregon hatchery intake. In both cases partial occlusion of the intake permitted sufficient air to be drawn into the systems to produce supersaturation and gas bubble disease in exposed fish. Hughes (1968) encountered a supersaturation problem resulting from air leaks in a lobster hatchery water supply.

Such air leaks on the low pressure side of a water supply system can easily cause supersaturation in a fish culture facility. This should be one of the first sources investigated when supersaturation problems are encountered in a pumped water source.

Johnson (1976) reported a somewhat different source of supersaturation. Seawater was pumped into an unused line that was filled with air. The pressure in the line caused the air to supersaturate the water sufficiently to produce gas bubble disease in the blue crabs and several fish. This appears to be an unusual cause of supersaturation but one that could easily be encountered in any piped water supply.

Fast et al. (1975) and Fast (1979) reported dissolved nitrogen supersaturation in New York and California lakes. In both cases, artificial aeration caused dissolved nitrogen concentrations to reach 140–150% of saturation.

Hydroelectric Projects

Spillways of hydroelectric projects cause air and water to be mixed and carried to substantial depths in a plunge basin. At the depths nor-

mally encountered in plunge basins, the hydrostatic pressure is sufficient to greatly increase the solubilities of atmospheric gases. The air thus passes into solution in sufficient amounts to produce supersaturation with respect to surface or atmospheric pressure. These sources of supersaturation frequently have the capacity to supersaturate large volumes of water and thus cause a major problem.

Jarnefelt (1948) recognized supersaturation associated with a hydroelectric project and measured oxygen supersaturation as high as 127% at a Swedish project. Total dissolved gas pressures were likely at least as high. Lindroth (1957) also measured supersaturation below a dam on the Indalsalven River, Sweden.

High levels of supersaturation in the Columbia and Snake rivers were first reported by Ebel (1969). During the 1966 spill period (July and August), nitrogen levels above 120% saturation were measured in the Columbia River. Dissolved gas levels remained near saturation during the remainder of 1966. In 1967, Columbia River saturation levels were comparable to those of 1966. Diurnal variations in nitrogen saturations were measured at The Dalles Dam during 1966; dissolved nitrogen concentrations were found to vary only 0.6 mg/liter during a 24-hour period of constant spill.

The effect of John Day Dam on the dissolved nitrogen levels in the Columbia River during 1968 was described by Beiningen and Ebel (1970). The dam was closed in April 1968, before the generators were operational, so that the reservoir could be filled and the fishways put into operation to accommodate the spring salmon migrations. This required total spill of all water passing John Day Dam and produced dissolved gas levels from 120% to 145% of saturation. Levels in excess of 125% of saturation were recorded below John Day Dam through September 1968. Water temperatures also increased due to the warming of water in the newly created John Day Reservoir.

Dissolved gas levels and associated variables for numerous locations on the Columbia and Snake rivers are presented by Beiningen and Ebel (1970) for the years 1965–1969. Ebel (1971) presented additional data for the year 1970. Dissolved gas levels were generally lower in 1970 than in previous years for the Columbia River but were high in the Snake River due to the spill at Little Goose Dam.

Roesner and Norton (1971) developed a mathematical model to evaluate air entrainment in water passing over spillways. The model was developed from monitoring data collected at three lower Columbia River dams (John Day, The Dalles, and Bonneville).

Meekin and Allen (1974a) reported dissolved gas levels associated with controlled-flow studies at Chief Joseph Dam on the Columbia River. Widely varying dissolved gas levels were measured in the upstream reservoir, as well as downstream, between individual controlled-flow tests. The reported variations within short time periods (2 hours or less) have not been found in other dissolved-gas monitoring studies. Although the report indicates the variations in supersaturation are probably due to different blocks of water, this conclusion does not appear to be supported by the data presented in the report.

Meekin and Allen (1974b) presented the results of dissolved gas monitoring in the mid-Columbia River from 1965 and 1971. Supersaturation occurred throughout this region of the Columbia River during high-flow periods. Grand Coulee Dam produced high levels of supersaturation, and the dams below Grand Coulee in this region increased the levels of supersaturation only slightly over those in water arriving at each dam. Priest Rapids and Rocky Reach dams actually reduced supersaturation when the water arriving in their forebays was highly supersaturated.

Meekin and Allen (1974b) also monitored the river upstream of Grand Coulee Dam in 1965, 1970, and 1971. During these years, this water was supersaturated to some degree. It exceeded 120% TGP in 1970 and approached this level in 1971. Seattle Marine Laboratories (1972b, 1974) monitored dissolved gas levels above and below Grand Coulee Dam in 1972 and 1973. Lake Roosevelt, the reservoir behind Grand Coulee, contained water supersaturated to over 120% TGP. This supersaturation was present in Columbia River water entering the United States from Canada. Downstream from Grand Coulee Dam, the Columbia River water exceeded 140% TGP in 1972.

Blahm (1974) and Blahm et al. (1975) described dissolved gas monitoring below Bonneville Dam on the lower Columbia River in 1974. Dissolved gas levels averaged near or above 120% TGP in this lower reach of the river dur-

ing May and June. The reports also described changes in dissolved gas concentrations as the river water traveled 110 km downstream from Bonneville Dam. The United States Army Corps of Engineers (1975, 1977) provided complete reports for dissolved-gas monitoring data for the Columbia and lower Snake rivers in 1974, 1975, and 1976. Boyer (1974) presented a detailed analysis of the dams and the various physical factors involved in the supersaturation problem in the Columbia River system, a valuable resource to anyone dealing with supersaturation resulting from dams.

Seattle Marine Laboratories (1972a) presented results of dissolved-gas monitoring studies on the middle and upper reaches of the Snake River in Idaho. This major tributary to the Columbia River contains numerous dams. Dissolved gas levels in the upper reaches of the river remained below 110% TGP. Water below a series of three dams in Hells Canyon along the middle reach of the river exceeded 120% TGP at times. Downstream from the last dam in Hells Canyon, dissolved gas levels gradually decreased to below 110% TGP at the confluence of the Snake and Salmon rivers. Parametrix (1974) again measured high dissolved nitrogen concentrations below the Hells Canyon dams; however, total dissolved gas levels did not exceed 110% due to low oxygen concentrations.

Dissolved gas concentrations in the Canadian portion of the Columbia River, its tributaries, and several other rivers were reported by Clark and Regan (1973), Clark (1974, 1976), and Abelson (1975). These rivers frequently exceeded 110% TGP and the Columbia River exceeded 120% TGP at times.

Thermal Increases

In recent years, electrical generating facilities have been constructed that significantly raise the temperature of large volumes of water. These temperature increases frequently have been sufficient to cause supersaturation of the water. Supersaturation occurs because the solubility of a dissolved gas decreases as the temperature rises while the actual volume of gas dissolved remains the same.

Harvey (1967) reported that water in a Canadian lake became naturally supersaturated during the late spring and summer of 1961. Solar radiation increased the temperature of

the lake sufficiently to produce supersaturation of 110–120% TGP at the level of the lake's thermocline.

DeMont and Miller (1972), Adair and Hains (1974), Jensen (1974), and Miller (1974) discussed various aspects of supersaturation of cooling waters from three steam generating stations in North Carolina. Supersaturation in the heated effluent of the Marshal Steam Station exceeded 120% TGP and apparently reached 130% TGP at times. Two associated steam stations, Allen and Riverbend, also caused supersaturation of cooling waters but a low incidence of gas bubble disease. The Marshal Steam Station caused greater temperature increases and produced supersaturation at high levels over a longer period of time than the other two stations. The level of supersaturation was related to the depth of discharge outlets and volumes of flow.

Marcello and Fairbanks (1976) described a similar problem at the Pilgrim Nuclear Power Station on Cape Cod. Although only dissolved oxygen was measured, these measurements do indicate that total dissolved gas levels exceed 140% at times and were frequently above 120% TGP. Experience has shown that oxygen levels normally are equal to or lower than total dissolved gas levels unless supersaturation is caused primarily by photosynthetic activity. The high levels of supersaturation at the Pilgrim Station were caused by temperature increases of 13–25 C over ambient temperatures. Only slight decreases occurred in the volumes of dissolved gases as indicated by oxygen concentrations presented in the report.

Supersaturation has been caused in other situations where water has been heated for culture purposes. Shelford and Allee (1913) heated water in a closed system, which caused supersaturation. The temperature increases of 8–17 C, without loss of dissolved gases, produced supersaturation sufficient to cause gas bubble disease. These temperature increases would have produced supersaturations of 115–130% TGP if the water had been saturated prior to heating and no gas was allowed to escape.

Emboly (1934) heated water in a closed system to hatch trout eggs. Temperature increases over 5 C caused gas bubble disease in hatching fry. A temperature increase in this range probably caused supersaturation of 112% TGP as

the water supply was apparently near saturation prior to being heated.

Erdman (1961) reported the disease in Atlantic salmon fry in stream water heated more than 2.8 C. The cold stream water was near saturation prior to the temperature rise. No dissolved gas levels were reported for the gas bubble disease incident. Stickney (1968) reported that Gulf of Maine waters were supersaturated due to photosynthesis and heating. Nitrogen levels as high as 128% were apparently due to temperature increases. Total gas pressures as high as 120% apparently caused no problem in natural waters but did cause the disease in fish when the water was pumped into the laboratory.

Lightner et al. (1974) encountered supersaturation in a heated seawater supply that caused gas bubble disease in brown shrimp. This seawater was heated from 22 C to 28.8 C in a closed system. The 7 C increase would have produced a supersaturation of about 112% TGP if the water were saturated prior to being heated.

Zirges and Curtis (1975) raised the temperature 4.5 C for the water supply to a chinook salmon hatching facility. Supersaturation was high enough to cause gas bubble disease in sac fry. Dissolved gas levels were reported to be 106% for oxygen and 190% for nitrogen. The 190% nitrogen figure appears to have been a misprint as the nitrogen level would only have increased to 110% due to the temperature rise if the water had been saturated prior to heating.

The episodes of supersaturation reported above indicate that supersaturation should be considered any time aquatic organisms may be exposed to heated water. This includes cooling waters of large industrial facilities or waters heated for aquatic culture purposes.

Natural Causes

Supersaturation is not a new or necessarily a human-caused phenomenon. Spring and well waters are frequently supersaturated with dissolved nitrogen although oxygen levels are frequently low. Marsh and Gorham (1905) discussed such situations in the well water supplies at a Tennessee and a New Hampshire fish hatchery. The aspirating effect of water flowing downward into aquifers carries air with it. The air is thus mixed with water under considerable

pressure in the aquifers. Oxygen concentrations may be reduced prior to use of these waters; however, the inert dissolved nitrogen gas remains in solution, causing supersaturation problems.

Marsh (1910) described a supersaturated well supply for a fish culture station along the Potomac River where dissolved nitrogen levels exceeded 140% of saturation. Rucker and Tuttle (1948) described a Washington State hatchery water supply having 110% TGP (120% nitrogen, 80% oxygen). Matsue et al. (1953) discussed a variety of artesian wells and springs in Japan that produced supersaturated water. Dissolved nitrogen saturations as high as 150–160% were reported.

Flowing surface waters may also be supersaturated by natural causes. Jarnefelt (1948) described how rapids can cause air entrainment and supersaturation just as dam spillways do. Ebeling (1954) and Höll (1955) both recorded oxygen supersaturation of streams, apparently due to falls and rapids. Mortimer (1956) described how bubbling and violent agitation, as in a turbulent stream flow, can produce supersaturation as well as reduce it. Lindroth (1957) observed that falls with deep plunge basins produce supersaturation while those with shallow basins at their base produce little or no supersaturation. Harvey and Cooper (1962) observed similar situations in a British Columbia coastal stream. Supersaturation occurred at high river flows with high falls having deep plunge basins.

Parametrix (1974) reported the Salmon River contained dissolved nitrogen levels as high as 121%; however, total dissolved gas levels seldom exceeded 110%. Even these levels of supersaturation are high when it is considered that river flows were extremely low in 1973, the year of monitoring. Supersaturation was the result of both temperature increases and slight increases in dissolved nitrogen concentrations as the water moved downstream. The increases in the amount of dissolved nitrogen were the primary factor producing supersaturation during the higher flow periods.

As discussed above under the topic of oxygen as a cause of gas bubble disease, photosynthesis can lead to high levels of dissolved oxygen in natural waters. Woodbury (1941), Alikunhi et al. (1951), Schmassmann (1951), Rukavina and Varenika (1956), Renfro (1963), and Supplee and Lightner (1976) all recorded cases of su-

persaturation caused by photosynthesis. It is likely temperature also played a role in these cases as the intense sunlight necessary for high levels of oxygen production through photosynthesis would also cause significant increases of the water temperature.

One case of natural supersaturation caused by geothermal heating has been reported (Bouck 1976). Total gas pressures of 107–110% were measured in streams heated by geothermal action in Oregon. Although 105% TGP was sufficient to cause hatchery trout fry to develop gas bubble disease, fish in the natural stream with 107–110% TGP showed no signs of the disease. This is a minor but obvious demonstration of the differences between natural and artificial situations and the difficulty that can be encountered in assuming the two are equally susceptible to any perturbation.

Solutions to Supersaturation

Supersaturation of water with dissolved gas results in an unstable condition that tends to return to a state of equilibrium. The rate at which this naturally occurs is usually too slow to prevent the problems discussed in this review. It is important to recognize that water is supersaturated only with respect to atmospheric or surface conditions. Hydrostatic pressure may greatly reduce or eliminate the tendency of the large volume of deeper water to equilibrate with respect to surface pressures. Deeper waters in reservoirs, rivers, oceans, et cetera, may actually have no tendency to lose dissolved gases as they are not truly supersaturated. The hydrostatic pressure that frequently causes the air to become dissolved at high concentrations will also maintain the high concentrations in subsurface water.

A variety of solutions to supersaturation in artificial water supplies have been used over the years. Marsh (1910) flowed water through shallow troughs with rough bottoms and over stacks of six perforated pans to reduce supersaturation. Both of these systems worked for small water flows. Embody (1934) was able to remove supersaturation by passing the water over a series of baffles placed at the head of a trough. Rucker and Tuttle (1948) succeeded in reducing dissolved nitrogen from 140% to near saturation by cascading water through a series of six troughs or shelves about 0.25 m apart. Harvey and Cooper (1962) used a splash tower with

12 sets of baffles to reduce nitrogen from near 120% to near saturation.

Dennison and Marchyshyn (1973) described a small inexpensive box designed to equilibrate water. The water flows in a thin layer over a perforated plate through which air is pumped to strip excess gases from the water. This device reduced oxygen saturations from 118% to 100%. Wold (1973) described the use of a large aerator facility for removal of excess dissolved gases at the Dworshak National Fish Hatchery in Idaho. These large agitators were able to reduce dissolved nitrogen levels from 130% to less than 105%.

Removal of excess dissolved gases in large bodies of water is considerably more difficult due to the stability of the supersaturated gases under many conditions. Harvey (1961) found that solar heating and mixing caused nitrogen supersaturation in lake water to a depth of 6–15 m. This supersaturated water mass remained fairly stable in volume and gas content from June through August. Rapidly flowing turbulent streams do not necessarily provide rapid equilibration of dissolved gases. As Mortimer (1956) pointed out, rapidly flowing streams may even produce supersaturation, and once supersaturated, water reaches equilibrium with the atmosphere slowly.

Ebel (1969) reported that no equilibration occurs in the five reservoirs between Chief Joseph and McNary dams (400 km) on the Columbia River during the high-flow period. Failure of the supersaturated water to equilibrate was blamed on lack of circulation and warming of surface water. When water is warmed, the decrease in the water's capacity to hold dissolved gas may compensate for any loss in the actual dissolved gas content and thus a high level of supersaturation will be maintained.

Beiningen and Ebel (1970) also discussed equilibration of supersaturated Columbia River water. They found that supersaturated water equilibrated to a greater degree in John Day Reservoir than in other previously studied reservoirs in 1968. This equilibration was thought to be due to the reservoir's great length and long retention time as compared to that of many of the other reservoirs.

Lake Roosevelt provided only slight equilibration of the supersaturated water that enters from Canada and travels 225 km to Grand Coulee Dam (Seattle Marine Laboratories

1972b, 1974). During summer months, the surface water of this reservoir tends to equilibrate in the downstream portion of the reservoir. The deeper waters apparently moving through the reservoir show little loss of dissolved gas.

The various mechanical solutions to the problem of supersaturation were discussed by Smith (1972). Perforated bulkheads to be placed in skeleton turbine bays have been built and tested. These reduced supersaturation but were detrimental to fish passing through the orifices. Long and Ossiander (1974), Long et al. (1975), and United States Army Corps of Engineers (1979) described tests to evaluate the effect of perforated bulkheads on juvenile salmon. Perforated bulkheads have also been proposed as a device for reducing the effective hydraulic head for operational turbines. Reducing the effective head would permit the passage of large volumes of water with minimum power generation. This procedure may be effective during the spring high-flow period when power requirements are about one-half of the maximum capacity of the major dams on the Columbia River.

Flip lips or spillway deflectors offer the potential of reducing the level of supersaturation produced by water passing over a spillway. These devices are structural modifications to the downstream face of a spillway that direct the spilled flow along the surface of the tailwater rather than allowing it to plunge to the bottom of the stilling basin. Tervooren (1972, 1973) reported the first results of tests performed on spillway deflectors installed at Bonneville Dam. These indicated that, under varying water conditions, deflectors would reduce the normal increase of excess dissolved gas in the spillway tailwater by 50%. These results were obtained when the forebay gas levels were between 100% and 120% saturation. This means that the installation of spillway deflectors for all bays at Bonneville Dam would result in downstream gas levels of 110% of saturation if forebay water did not exceed 100% of saturation.

Spillway deflectors are effective in reducing supersaturation without causing increased mortalities of juvenile salmonids. Johnson and Dawley (1974) reported that the Bonneville deflectors reduced supersaturation by 6–12% with no decrease in the survival of fish passing over them; Monan and Liscom (1975) actually found

improved adult survival. Long et al. (1975) also reported an increased survival of steelhead smolts passing over deflectors at Lower Monumental Dam on the lower Snake River.

The recent status of spillway deflectors has been reviewed by United States Army Corps of Engineers (1979). Ebel et al. (1975) estimated that the installation of recommended deflectors and turbines by 1980 will eliminate the problem of supersaturation in the Columbia and Snake rivers for all practical purposes. Ebel (1979) concluded the supersaturation problem in the Columbia system has been solved.

A third proposed solution to the supersaturation in the Columbia River system is the collection and transportation of juvenile salmonids from upstream dams to the lower river (Ebel et al. 1973; Ebel et al. 1975). Although the supersaturation problem is apparently being solved by other means, this method is still being utilized to reduce the turbine mortalities that are considerably greater than supersaturation mortalities.

Supersaturation problems caused by cooling waters of power generating facilities have been less of a problem than hydroelectric projects and therefore have received far less attention. Kraback and Marcello (1976) discussed the feasibility of removing excess dissolved gases from the Pilgrim Nuclear Power Station cooling water. Heated seawater would be degassed by means of an air bubbler system. Tests in a flume installed in the discharge canal indicated that the system would work.

Lee and Martin (1975) computed the capacities of high- and low-velocity discharges of cooling to produce supersaturation problems. They concluded that high-velocity discharges are unlikely to cause supersaturation problems because of rapid dilution. Localized supersaturation problems may occur with low-velocity discharges.

Dissolved Gas Analysis

The measurement of dissolved gas levels is the one aspect of supersaturation that is least familiar to most people who must deal with the problem of gas bubble disease. Analysis of the levels of supersaturation is, however, essential for the evaluation and solution of the supersaturation problem. Although most people likely to encounter supersaturation are familiar with dissolved oxygen analysis, few have had

any reason to become familiar with the techniques for dissolved nitrogen determination or of total dissolved gas analysis. This section of the review describes a few of the more recent reports that discuss useful information dealing with such analyses.

The solubility of the individual atmospheric gases in water has been described in numerous recent reports. Elmore and Hayes (1960), Klotts and Benson (1963), Green and Carritt (1967), Douglas (1964, 1965), Murray et al. (1969), and Tolk et al. (1969) all discussed the solubilities of oxygen, nitrogen, and argon. The solubilities most commonly used in recent supersaturation research are those provided by Weiss (1970).

There are several different techniques available for dissolved gas analysis. Swinnerton (1962) described the use of gas chromatography for this purpose. Beinigen (1973) provided a complete discussion of the use of the time-consuming Van Slyke apparatus (Oesting 1934) for determining the oxygen and nitrogen-plus-argon concentrations of water samples. Beinigen discussed in easy-to-follow detail the operation of this apparatus as well as the calculations required for the separate dissolved gas analyses. He included sections on the correct procedures for field sampling, procurement of the necessary apparatus, and the Winkler method of oxygen determination. This manual is extremely useful for anyone using the Van Slyke method to determine dissolved gas concentrations in water samples.

Post (1970) described a simplified volumetric method for determining dissolved gas concentrations. This method apparently has received little use and may provide significant errors of 5–7%.

By far the simplest method for determination of total dissolved gas levels is that provided by the Weiss saturometer. As far as can be determined, this apparatus has not been described in any journal or other widely distributed publication. Fickeisen et al. (1975) gave a brief description and picture of a Weiss saturometer. A similar device is produced commercially by ECO Enterprises, Seattle, Washington, and is now widely used to measure total dissolved gas pressure.

D'Aoust and Smith (1974) and D'Aoust et al. (1976) described a modification (tensionometer) of the Weiss saturometer. The tensiono-

meter provides for sensing of the gas pressure by means of a solid state electronic pressure transducer rather than the Bourden tube gauge used in the saturometer. According to D'Aoust et al., the tensionometer has the advantages of much smaller size, and a response time of about 8 minutes. This compares with a response time of 20–30 minutes for the saturometer. The more rapid response time of the tensionometer is due to the much smaller dead space permitted with the pressure transducer. The tensionometer also offers the advantage of remote sensing which is required when dissolved gas measurements are made at depth. Agitation is still required to remove bubbles from the silastic tubing when the water is truly supersaturated, such as at surface pressures of about one atmosphere. This is not a problem at water depths over 3 m where the water is not actually supersaturated due to the increased pressure of the hydrostatic head. D'Aoust et al. (1976) provided a complete parts list and instructions for building the tensionometer.

The operation of the Weiss saturometer was evaluated by Fickeisen et al. (1975). The saturometer was mechanically agitated at rates of 108, 132, and 168 cycles per minute. At the two faster rates, equilibrium was reached in 15–25 minutes. It requires 10–15 minutes longer to reach equilibrium at the slower rate of agitation. Manual operation produced comparable results with an experienced operator but lower readings with a novice operator.

Jenkins (1976) attempted to develop a method for unattended monitoring "in situ" of dissolved gas concentrations. An incomplete system was designed to pump the water to be sampled and to strip the dissolved gases from this water by a modified spinning disc oxygenator. Major problems remain to be solved in the detection of the gases once they are stripped from the sample water.

The various corrections that have been applied to dissolved nitrogen partial pressure data were discussed by Boyer (1974). He concluded that the sum total of these corrections is less than the inherent errors of the sampling and analysis techniques. Boyer also discussed the "true" value of the supersaturation level resulting from a dam spillway and described what is needed to develop an empirical formula that is unique for each spillway.

Cratin et al. (1971) studied the in situ fixation

and analysis of dissolved oxygen samples at depth using an underwater habitat (Teküte II). In marine waters, they found a decrease of about 2–6% in the dissolved oxygen concentration of samples fixed at the surface as compared to those fixed at depth. This loss of oxygen was limited to samples whose oxygen concentrations were greater than the surface saturation value. The loss of dissolved oxygen should not be a problem with supersaturation monitoring as the few deep samples collected are normally fixed immediately after collection. There is no indication of how dissolved nitrogen concentrations vary prior to analysis by Van Slyke or gas chromatograph. The amount of dissolved gas these samples can hold is normally increased during storage by reducing the temperature of the samples to below 4 C. Thus, supersaturation in the samples is reduced or eliminated, preventing loss of dissolved nitrogen.

Regulation of Supersaturation

Since the identification of dissolved gas supersaturation as a problem in the Columbia River system in the late 1960's, there have been criteria and standards promulgated by a variety of regulatory entities. The National Academy of Sciences/National Academy of Engineering (1972), using available data, recommended that aquatic life will be protected when total dissolved gas pressure in water is no greater than 110%. Subsequently the states of Washington, Idaho, and Oregon promulgated dissolved gas standards, initially for dissolved nitrogen and later for total dissolved gas. The regulations specified human activities should not increase dissolved gas levels above 110% in Washington and Idaho and 105% in Oregon. Other states have since passed similar regulations.

The water quality standards were reviewed in 1975 by a group of four agency representatives from the United States Environmental Protection Agency, Idaho, Oregon, and Washington (Rulifson and Pine 1976). This group suggested a standard of 115% total gas saturation for the Columbia-Snake River system except during particularly high-flow years. Their recommendation was ignored by the Environmental Protection Agency criterion issued in 1976 (USEPA 1976), which again recommended a criterion of 110% TGP. Recently, Ebel et al. (1979) have reviewed the most recent Environmental Protection Agency criterion, and in-

dicating defensible dissolved gas criteria could be established at either 110, 115, or 120%.

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